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G-INDUCED LOSS OF CONSCIOUSNESS AND ITS PREVENTION

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19. ABSTRACT (Continue on reverse if necessary and identify by block number)

The current capabilities of trained individuals to maintain clear vision during sustained exposures to 9 G_z, an increase in protected G tolerance of about 4 G over World War II fighter pilots, is a result of combined use of a G suit and very effective self-protective straining maneuvers such as the M-1, L-1 and pressure breathing, all of which are variants of the Valsalva maneuver developed in the 1940s. However, more than ten fatal crashes attributed to acceleration-induced loss of consciousness have occurred in recent years. The most plausible causes are: (1) increased capability of jet-powered fighters to sustain, with minimal pilot effort, accelerations in the 7-10 G_z range for periods longer than the symptom-free 3-8 second cerebral ischemic anoxic period which precedes GLOC, (2) an improperly performed Valsalva-type straining maneuver, and (3) development of a hypotensive vasovagal type reaction. A foolproof GLOC warning system such as detection of zero arterial pulsations at ear level to activate an automatic plane control takeover system could avoid most GLOC crashes. However, an omni-directional surveillance fighter plane cockpit for a fully

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Valsalva-type maneuvers
Intrarectal (intra-abdominal) pressures
Airway and intra-abdominal pressure relationships
Physiology of self-protective maneuvers
Single pressure and arterial occlusive anti-G suits and valves
Vasovagal reactions
Petechial hemorrhages
Limitations and dangers of high-G protection

19. ABSTRACT (Continued)

horizontal position, preferably prone, is the only physically and physiologically
certain and safest way to eliminate GLOC and to achieve the probable tactical
advantages of sustained combat maneuvering in the 9-15 G_z range. *Keywords)*

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List of Figures

G-INDUCED LOSS OF CONSCIOUSNESS AND ITS PREVENTION
W.K. STEWART MEMORIAL LECTURE*

Air Marshal Hurrell, President of the Royal Aeronautical Society, Air Commodore Ernsting, President of the Aviation Medicine Group, Stewart Trustees, Ladies and Gentlemen, and especially Bill Stewart's widow, Lady Brian Matthews.

It is for me a real privilege and a great honor to be invited to give the Stewart Memorial Lecture for 1987.

I will not attempt to recount the unique and exceptional accomplishments of the very charming and strong personality who was Bill Stewart. A much better alternative is to suggest that any of you who haven't done so, experience the inspiration which quite certainly one feels when reading the concise account of his career contained in the Stewart Memorial Prize Booklet (1). Rather, this account will be limited to my own in-person contacts with Stewart, which possibly may be of historic interest to some of you.

Our first meeting occurred in 1944 when he visited the Mayo Aero-Medical Unit in Rochester, Minnesota, to review the intensive wartime high-altitude decompression chamber studies on oxygen masks, use of denitrogenation for prevention of bends and chokes, and related activities; but, especially to see and test for himself on the Mayo Human Centrifuge, the then new, simplified single pressure anti-G suits and valve systems, the very effective so-called M-1 self-protective maneuver, plus the objective physiologic instrumentation which had been developed to study the physiology of blackout and unconsciousness in human subjects and means for their prevention, both on the human centrifuge and in a specially instrumented dive bomber in flight.

Stewart's intense dedication relative to the blackout problem in fighter pilots was evidenced by the fact that he spent much of one day undergoing multiple high-G exposures on the centrifuge with and without the use of various anti-blackout suits and protective maneuvers.

My next meeting with Bill was at the Institute of Aviation Medicine in Farnborough in the summer of 1946 on my return from a 3-month period as a scientific consultant at the U.S. Air Force Aero-Medical Center which had been set up at the former Kaiser Wilhelm Institute in Heidelberg, Germany (2). Our mission was headed by Dr. E.J. Baldes, who was one of my mentors at that time and who, by the way, received his doctorate degree in biophysics at University College, London, under A.V. Hill. Our orders as consultants of the then U.S. Air Surgeon, General Malcolm Grow, were to interview individuals, in Germany, who had been involved in aviation medicine before and during World War II, and persuade and arrange for each to prepare a review of the particular aspect of aviation medicine in their special area of expertise. Because of the chaotic situation in Germany at that time, they accepted this

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opportunity with enthusiasm and the subsequently published, quite impressive, two-volume review of German aviation medicine during World War II, resulted (3).

Characteristic of Bill Stewart's friendly, hospitable personality, he invited me to accompany him to a military ball at Aldershot along with his gracious wife and another couple from Farnborough. This period was shortly after the time your so-called "tight little island" had been somewhat overrun with Yankee G.I. soldiers who, perhaps to some degree justifiably, were characterized by some of your countrymen as being "overpaid, oversexed, and over here."

This military ball perhaps was the background for the most gracious, but deeply rooted, put-down I have ever experienced. This put-down occurred while sitting out a dance with the wife of one of Stewart's colleagues. In a most friendly manner, she asked me: "How do you like England?" In an attempt to be friendly in kind, I replied: "I like England and the English very much. After all, by descent, I am mostly English myself." She then replied, without apparent malicious intent, "Oh really, isn't it amazing how many Americans like to think they're English?"

In this regard, I can't resist outlining my own quite definite English connection. My mother's name was Inez Goff spelled GOFF. As many of you know, Goff, with a variety of spellings, is an old English name. An expert family geneologist has traced her roots back to one of the old, relative to America's brief history, New England Goff families and is certain that one of my great-great-great-uncles, William Goffe, was a general in Oliver Cromwell's Army who, because he was one of the appointed judges whose verdict caused your former King Charles I to be beheaded, subsequently became a fugitive from the King's men in the then American Colonies.

Figure 1 is a drawing of an incident in this regicide's life (4), who when leading a shadowy and perilous life on the then American frontier, came out of hiding to forewarn and then direct the defense of the small New England village of Hatfield, Massachusetts, from being massacred by hostile Indians. The rather fanatic looking character is William Goffe. Parenthetically, in today's parlance, the hostile Indians or "contras" would have quite certainly been called "freedom fighters," at least by the French authorities of those times.
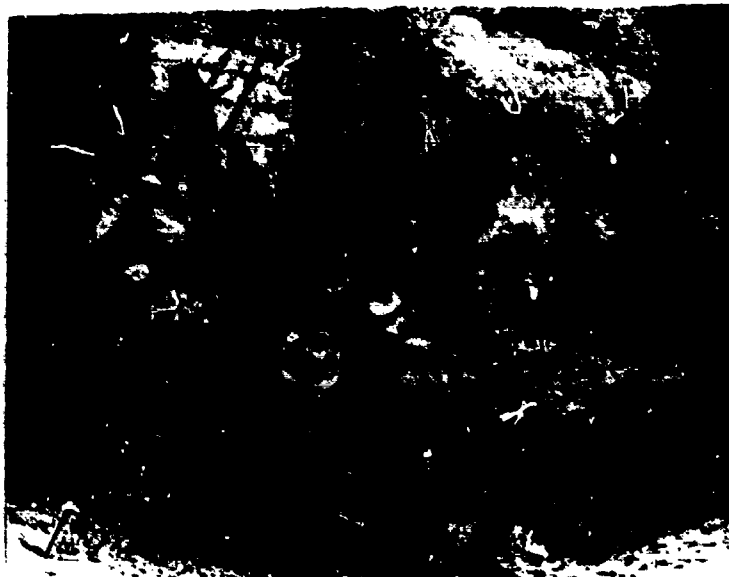
Figure 1. An imagined recreation of the fugitive regicide William Goffe directing the defense of Hatfield, Mass. against an attack by hostile Indians, Circa 1675. (From The John Goffe's Legacy [4]).

Now to the main content planned for this evening's discussion, which I forewarn you will consist of 4 segments of cinefilm interspersed with 39 slides and narrative for a total additional time of about 48 min. So, as they say in the airline business, sit back, relax, and enjoy the flight, which on this occasion will be a flight of facts and ideas which hopefully will be of some interest to some of you.

Positive G-induced loss of consciousness (GLOC) can be defined as a loss of cognitive brain function due to acute cerebral ischemic anoxia caused by exposure to an increased gravitational-inertial force environment sustained for longer than the 3-7 sec ischemic anoxic latent period between acute stoppage or near stoppage of brain blood flow and loss of cognitive brain function.

The hydrostatic basis and resulting biodynamics of loss of consciousness during sustained high-G aerial combat maneuvers have been increasingly well understood since before and during World War II (5-14). However, because of the very high G tolerance of current fighter pilots (15,16) made possible by supplementing the about 1-G protection afforded by the standard, relatively ineffective anti-G suit, concomitantly with very effective voluntary self-protective straining maneuvers, both of which were developed during World War II (17-19), GLOC had not been considered to be an operationally important problem until the mid 1980s (20-22).

The now quite wide-spread recognition of the pilot and plane destroying consequences of GLOC in today's very high speed, highly maneuverable fighter planes has been prompted by multiple GLOC-related fatal crashes (10-14, 23-26), one of which is illustrated in Figure 2.

Crash of F-20A Prototype
G-Induced Loss of Consciousness
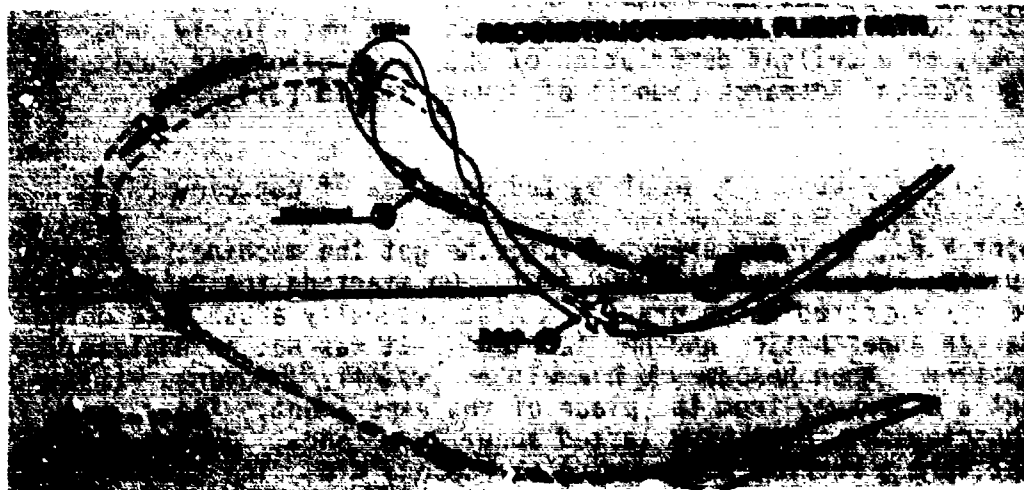
Figure 2. Reconstruction based on cinefilms of the final fatal flight path (solid lines) of a prototype F-20A Tigershark fighter plane. The dashed lines are the intended flight path which was to be a routine landing after a simulated head-on, low-level, air-to-ground attack followed by a sharp 7- to 9-G pull-up (26). (From Aviation Week, March 16, 1987).

3

This copy of the lead-off figure in the March 16, 1987 issue of Aviation Week (26) is a reconstruction based on cinefilms of the final fatal flight path of a prototype F-20A Tigershark fighter plane flown by Mr. David Barnes, a 40-year-old engineering test pilot wearing a properly fitted U.S. Air Force G-suit. This very experienced pilot had flown this same simulated head-on, low-level, air-to-ground, attack followed by a sharp 7- to 9-G pull-up about 60 times during prior months, and this flight was the fourth time that day.

The conclusion of a very thorough investigation of this crash by the Canadian Safety Board designated pilot loss of consciousness as the causative factor (26).

This fatal crash and a succession of at least ten other GLOC crashes during recent years (24,25), are the basis for the intense interest and ongoing, very active multifaceted investigations of the causes and means of preventing GLOC (10-14,24,25), which hopefully can be implemented as expeditiously as possible, to reduce the frequency and/or the consequences of this potentially fatal situation and ultimately, possibly over a longer time frame, actually eliminate GLOC (12,13) as a cause of incidents such as illustrated in Figure 2.

The physiology of acceleration-induced loss of consciousness, the understanding of which has been progressing since before World War II (5,12,13,27-29), indicates that GLOC-caused fatal crashes are preventable accidents. Increased understanding of this physiology offers the best hope for prevention of GLOC in the future.

There is firmly based evidence that G-induced zero or near zero arterial systolic blood pressure at brain level for periods of 5 or more seconds, and the consequent severe decrease or cessation of cerebral blood flow for like periods are the cause of GLOC (10,13,27-30). The following comments concern the historical development of this understanding.

Incidents of so-called fainting in the air were reported by the Royal Air Force during World War I; however, their cause was not clearly understood. Nevertheless, an excellent description of GLOC by a pilot was published in 1919 by The Medical Research Council of Great Britain (31).

An Early Description of G_z-Induced Loss of Consciousness

"Major V.B., a highly expert pilot...he got the machine, a Sopwith Triplane, into a turn at 3,000 feet. On starting the second turn 'the sky appeared to go grey'. A mist gradually arose like going under an anaesthetic, and he 'fainted'. It was not an unpleasant sensation. When he came to himself, he was flying over a village about a mile away from the place of the experiment. The unconsciousness must have lasted about 20 seconds. During the first turn g reached 4.5, during the second 4.6. The turn was of about 140 feet at a speed of 114 miles an hour (Figure 3)."

From: Head, Henry "The Sense of Stability and Balance in the Air."
In: The Medical Problems of Flying, pp. 215-254, May 1919.
Medical Research Council, London, England.

4

Figure 3. The British Sopwith triplane capable of sustaining accelerations of
4.5 G in spiral turns during which this incident of "fainting in
the air," presumably GLOC, occurred (31). (Picture courtesy of
Royal Aeronautical Society, London.)

The next quite accurate description of GLOC and its cause, from the same
hydrostatic considerations which are accepted today (13), was published in
Germany by von Diringshofen (Figure 4) in 1934 (5).

Figure 4. Dr. Heinz von Diringshofen established the world's first "flying"
aeromedical laboratory where, beginning in 1931, he measured the
effects of G forces. His brother Bernd, an engineer, helped deve-
lop airborne instrumentation. (From Harald von Beckh [32]).

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This description and his pioneering, quite accurate observations of blackout and losses of consciousness in volunteer subjects (Figure 5) during sustained high-G aerial maneuvers and particularly his in-flight measurements of arterial blood pressure, I believe, justify designating von Diringshofen as the father of hyper-gravitational physiology.
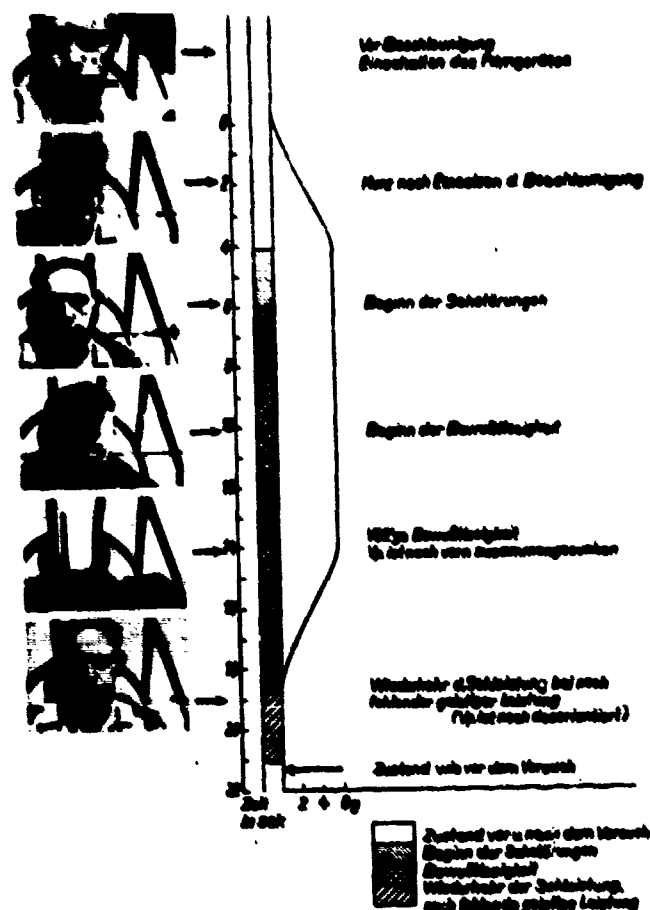
Figure 5.   Selected cine frames of an incident of loss of consciousness in flight (5). Note the delay ('atent period) of about 6 sec between attainment of 6 G and the resulting loss of consciousness indicated by involuntary bowing of the head and slumping forward in the seat. From "Grundris der Luftfahrtmedizin" edited by Siegfried Ruff and Hubertus Strughold, 1939 (33).

The in-flight, 1942-44, personal experiences and observations of blackout and GLOC, by Bill Stewart (whom we are honoring tonight) are the next easily recognizable milestone in hyper-G physiology, at least in the Allied Nations (7). As you all know, Stewart played the dominant role in Acceleration Physiology Research at the Institute of Aviation Medicine in Farnborough throughout World War II (1).

His observations can be most easily appreciated by cine filmstrips of him in the rear cockpit of a Fairy Battle aircraft. Selected frames from one of these filmstrips of Stewart experiencing an incident of GLOC indicate the hazardous nature of these studies (Figure 6).
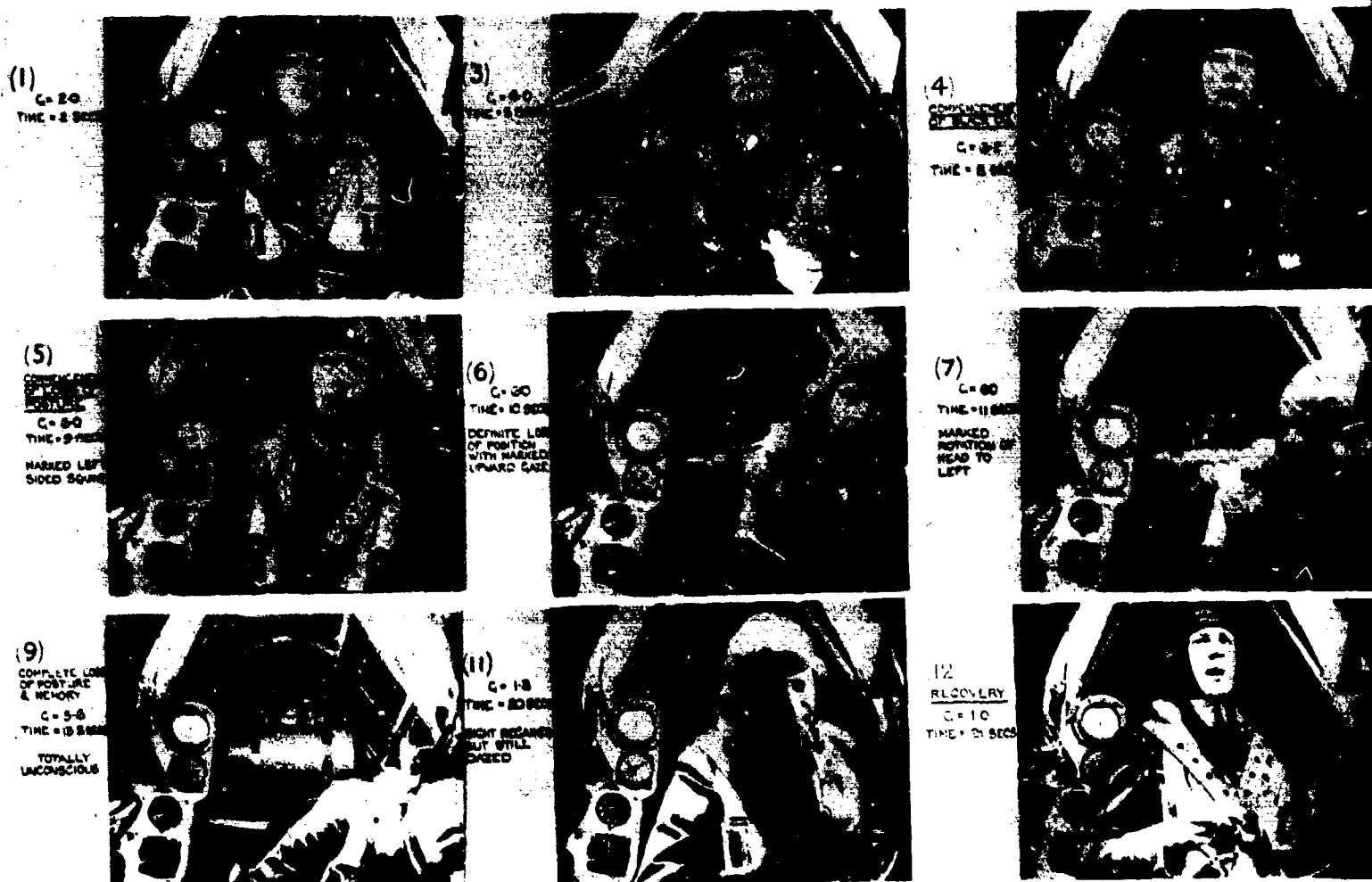
Figure 6. Selected frames of an in-flight incident of GLOC experienced by Dr. W.K. Stewart, RAF, Farnborough, England (reproduced from [7]).

Bill Stewart was forced to study the effects of acceleration in aircraft because, at that time, there was no human centrifuge in England.

The first human centrifuge in the Allied Nations capable of simulating the acceleration profiles of aerial combat maneuvers attained operational status in 1942 in Toronto, Canada (Figure 7) under the leadership of Wing Commander Bill Franks of the Canadian Air Force (8,34).

7
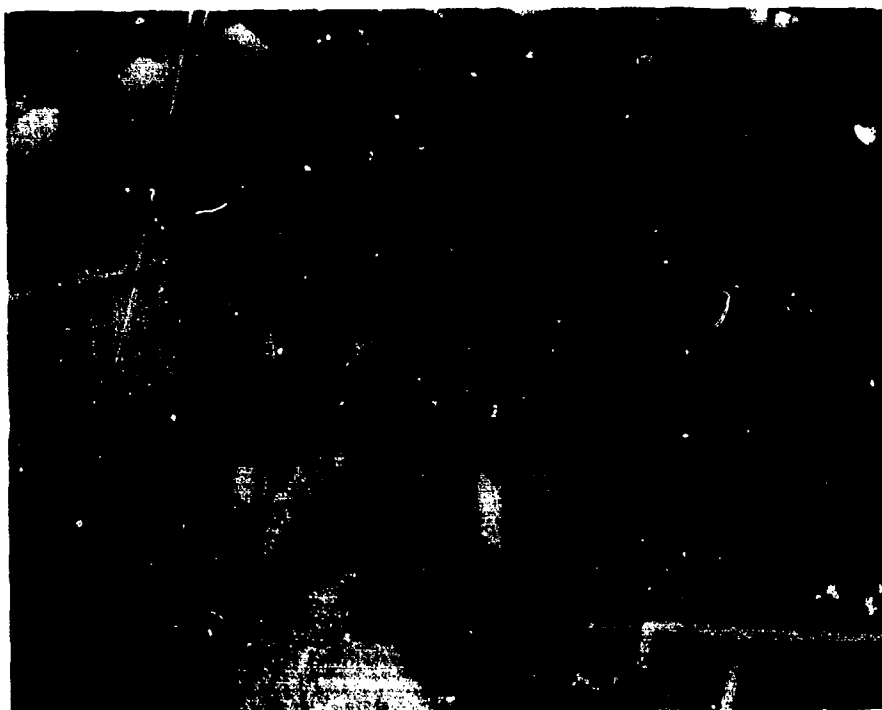
Figure 7. The first modern human centrifuge in the Allied Nations under the
          leadership of Wing Commander W. Franks, RCAF, Toronto, Canada,
          1942.

     The first, by several years. human centrifuge with these capabilities in
the United States was activated later that same year at the Mayo Clinic in
Rochester, Minnesota (Figure 8).
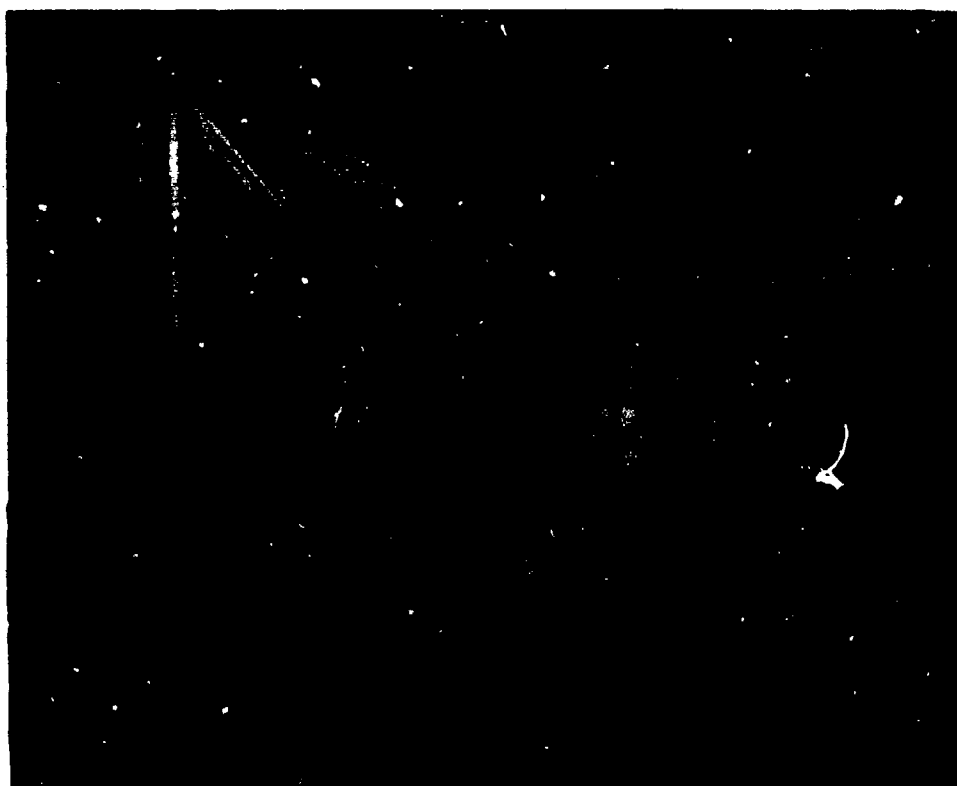
Figure 8.   Mayo Human Centrifuge (35,36). The cockpit, in the foreground, was
            free to swing radially during centrifuge rotation so that the
            resultant vector of the gravitational and inertial forces to which
            the subject is exposed is along the heart-to-brain axis (G_z
            acceleration). The observer controls the time of starting and
            stopping of the centrifuge and tests the subject's vision
            throughout the period of exposure by activating lights mounted on
            the subject's instrument panel in the cockpit at his fixation point
            (central vision) and 23° from his fixation point (peripheral
            vision). The subject's response times to these light signals and
            the alterations in other physiological parameters are transmitted
            electrically through a commutator system mounted on the center
            shaft of the centrifuge and recorded photokymographically in an
            adjacent room. See Figures 16 and 19 for an example of physiologi-
            cal recordings obtained with this assembly.

     The operation of this centrifuge and the objective instrumentation metho-
dology used for quantitative studies of the physiologic responses of healthy
humans to acceleration under laboratory conditions can be most easily
understood by films made on this machine and in a specially instrumented dive
bomber assigned to our laboratory by the U.S. Air Force (36-39). Figure 9 of
selected cine frames of a healthy male volunteer experiencing GLOC in an A-24
(Douglas Dauntless Dive Bomber) and the simultaneous physiologic recordings
illustrate the nature of the Mayo Human Centrifuge and in-flight studies
carried out during World War II.

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SUBJECT 26, UNPROTECTED, PASSENGER IN A-24 AIRPLANE
5.0 g
(Symptoms: "Blackout", Disorientation)
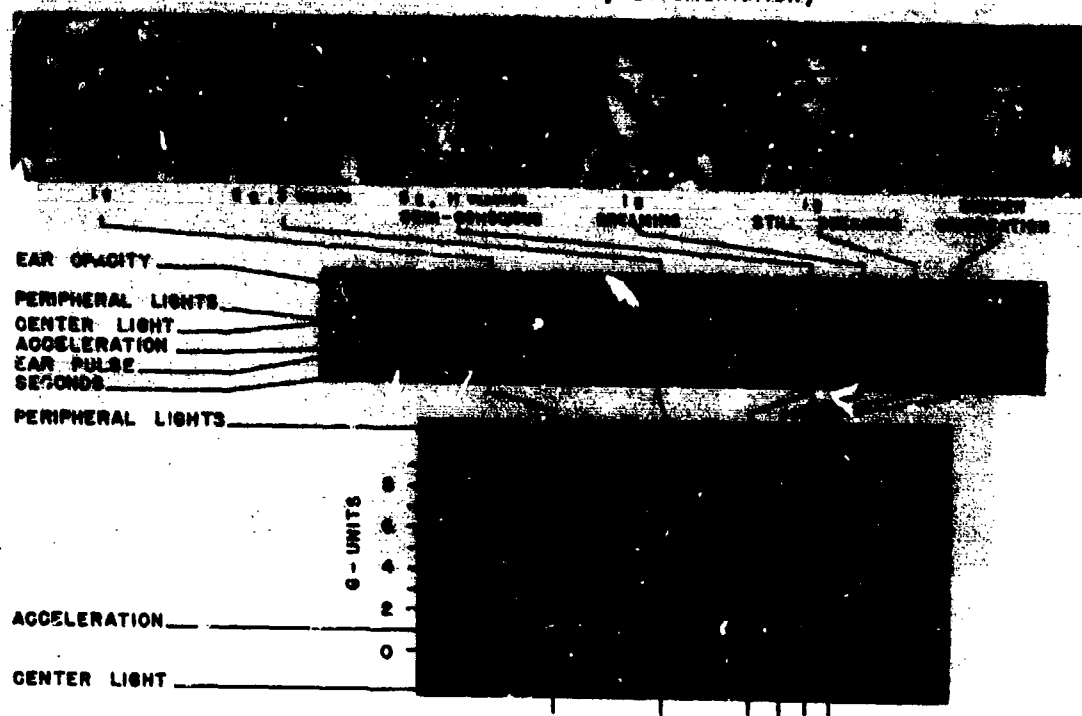
Figure 9. The effect of positive acceleration of 5.0 G on a passenger in an airplane. The photographs are enlargements from a 16-mm motion picture. The cap worn by the subject protects the photoelectric ear units from sunlight. The middle panel is a six-channel recording from a cathode-ray oscillograph. The lower panel is the acceleration vs. time curve and the subject's reaction times to peripheral and central light signals during this experiment. Black lines synchronize the motion pictures and other records. The subject stated that he blacked out in this run and was confused. Apparently, consciousness was impaired and he was disoriented for about 7 sec after return to 1 G. Note reduction of the ear pulse which preceded the losses of peripheral and central vision by several seconds and the apparent loss of consciousness by about 8 sec indicating that this objective parameter could be used as a forewarning of impending loss of vision and/or consciousness. The prolonged failure to respond to light signals is characteristic of the slow recovery from GLOC as contrasted to the rapid recovery of responses to light signals when blackout occurs without loss of consciousness (37) (from Lambert, E.H. [40]).

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The major accomplishment of the Mayo and other human centrifuges in the
Allied Nations which attained operational status during World War II was to
develop the objective instrumentation and associated techniques required to
understand the physiology of blackout and GLOC (41,42). These capabilities
provided the basis for development and quantitative documentation of the mode
of action and effectiveness of various protective stratagems (43). Of these,
the simplified, pilot acceptable, single pressure anti-blackout suits (44-46),
their inertially activated G-valve systems (47), the M-1 self-protective
maneuver (17,18), and variations thereof such as the L-1 (15) and pressure
breathing (48), are still tactically important today (12). However, the
potential tactical importance of other World War II developed, more effective,
protective stratagems such as more complete bodily coverage, higher pressure
anti-G suits (49,50), fully horizontal positions (12), and ear plethysmography
techniques that forewarn of impending blackout and/or GLOC (51-54) will
apparently not be recognized and utilized until when and if: (1) the total
elimination of GLOC during pilot training and combat maneuvers, and (2)
exploitation of the very high sustained G-maneuvering capabilities of piloted
current and future very high performance fighter planes are implemented as
high-priority objectives by the military.

Figure 10 is a picture of the simplified single pressure bladder system
which was the basis for the quite successful G-suits used during World War II
and up to the present. This system and a similar single pressure bladder
system used by the Royal Air Force (46) were incorporated into a variety of
close-fitting trouser or coverall type garments (Figure 11) which provided an
increase in G tolerance of about 1.5 G. However, because of convenience and
greater comfort, the externally worn, less effective cutaway garment (Figure
12) became the standard U.S. Air Force anti-G suit.

Figure 10. Simplified anti-G suit pneumatic bladder system (49). The abdomi-
            nal bladder (at the top) bilateral thigh (center), and calf bladders
            (bottom) were inflated to a single pressure via a G activated and
            compensated inertial control valve. This bladder system was
            incorporated into a variety of garments (Figure 11).

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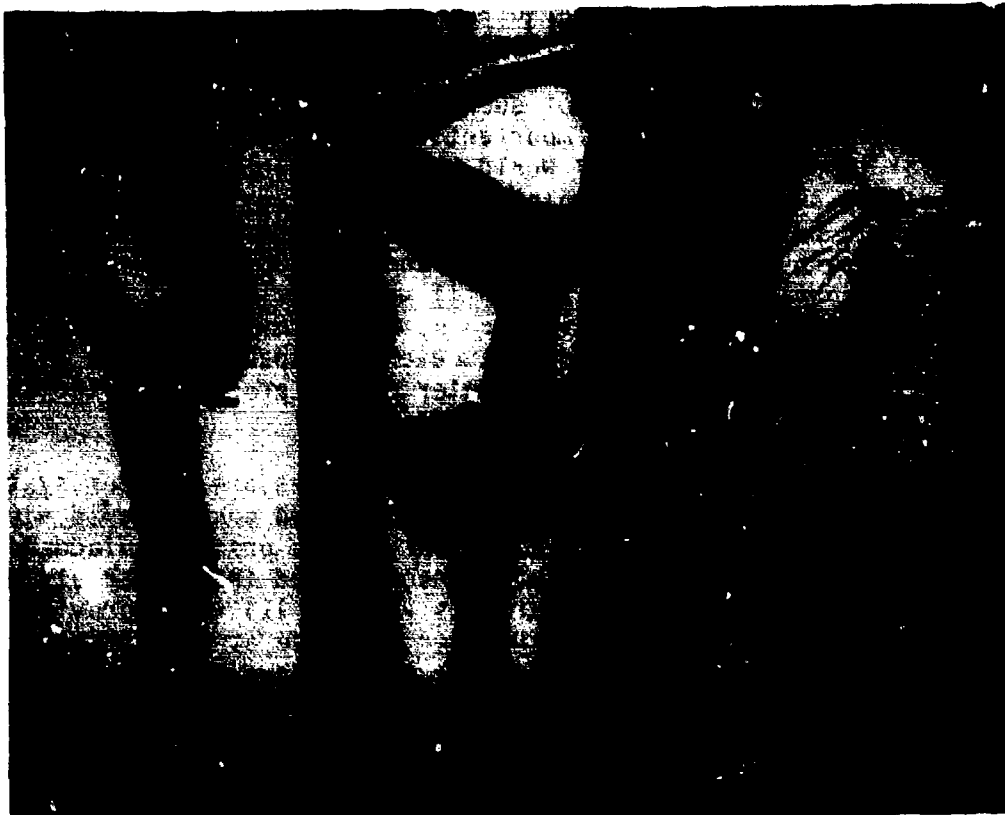
Figure 11. The bladder system (center panel) for simultaneous application of
           pressure to the calves, thighs, and abdomen, was incorporated into
           various types of coverall, trousers, and cutaway type garments
           that provideu relatively efficient transmission of the bladder
           pressure to the desired surfaces of the body. All operationally
           used anti-blackout suits, of which these are precursors, are close-
           ly similar to the system illustrated. The full coverall suit on
           the right was used during World War II by U.S. Navy pilots. This
           system was devised by Mr. David M. Clark, formerly of Worcester,
           Massachusetts. Historical reviews of the development of anti-
           blackout suits prior to and during World War II have been prepared
           by Thomas W. Walker in "Blackout! The Development of the Anti "G"
           Suit. "G" Suit Pioneers in the U.S., Germany, Australia
           (1939-42)." Aerotec Industries Review, Autumn, 1959, pp. 4-6, and
           by Harrison and Gibson (46).

Figure 12. Cutaway or skeleton type anti-blackout suit with its inertially activated G-valve system. This suit was used by the U.S. Air Force during World War II and is still the G-suit most widely used by USAF fighter pilots.

The mode of action of all effective anti-G suits is to provide the transient hypertension at heart level required to overcome the increases in hydrostatic distance between heart and head level which, in the upright seated position, is an unavoidable consequence of exposure to acceleration (44,48,55).

The instrumentation used back in the 1940s to collect the data upon which this conclusion is based and some of the recordings obtained therefrom are illustrated in the next series of figures.

Development of objective methods to study hyper-G physiology and the mechanisms of action of anti-G suits and other protective maneuvers or devices, was of highest priority at that time. Since the decrease in blood pressure and consequent decreased circulation at head level are the most dangerously acute effects of headward acceleration, noninvasive objective methods related to blood flow at head level were of prime importance for studies of G-tolerance and the effectiveness of anti-G suits. Photoelectric plethysmography of the ear (Figure 13) proved to be excellent for these purposes (51-54). Two frames from a 1943 motion picture of a subject on the Mayo Human Centrifuge (Figure 14) obtained at 1 G (left panel) and 5 G (right panel) illustrate the mode of fixation of bilateral earpieces used for most volunteer subjects.
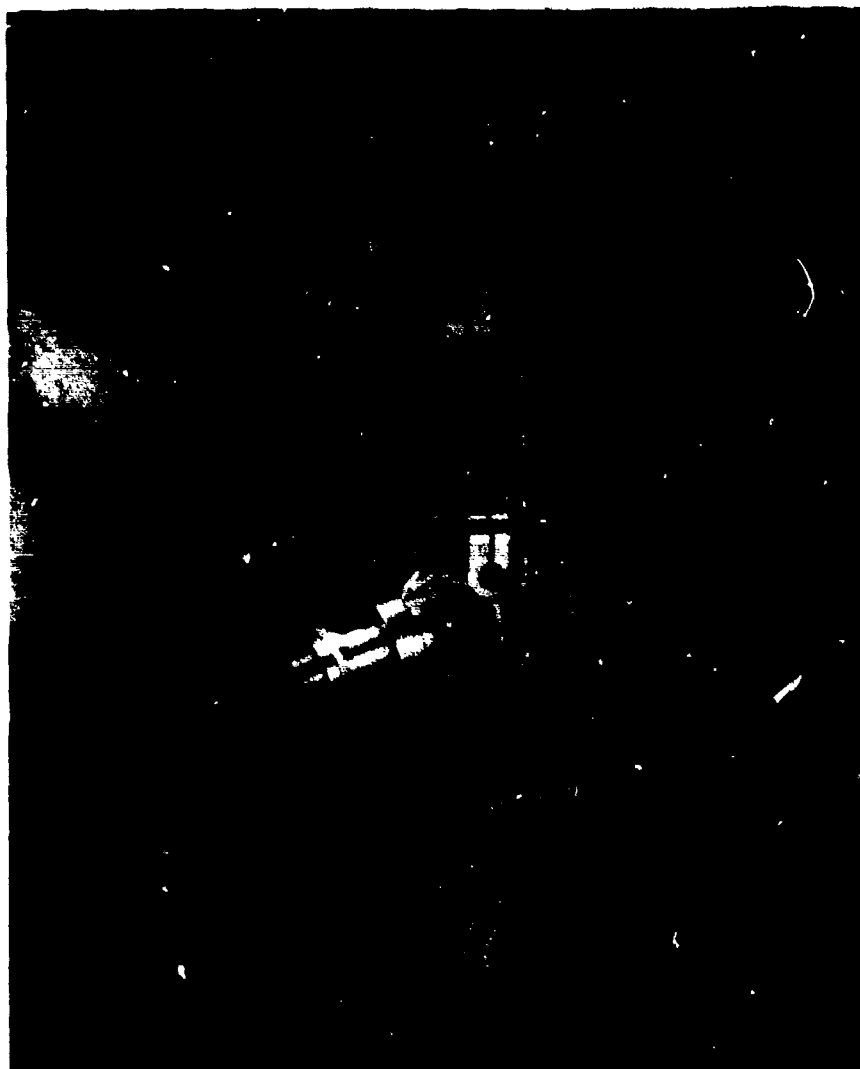
Figure 13. Photoelectric earpiece in place on the pinna of the ear and attached by means of a special universal clamp to a plaster helmet made individually for veteran subjects. This assembly was used to maintain the position of the earpiece which detected the variations in opacity of the ear associated with the changes in its blood content produced by exposure to acceleration (54). (Reproduced with permission from E.H. Wood et al., J. Applied Physiol., 1963, 18:1171.)

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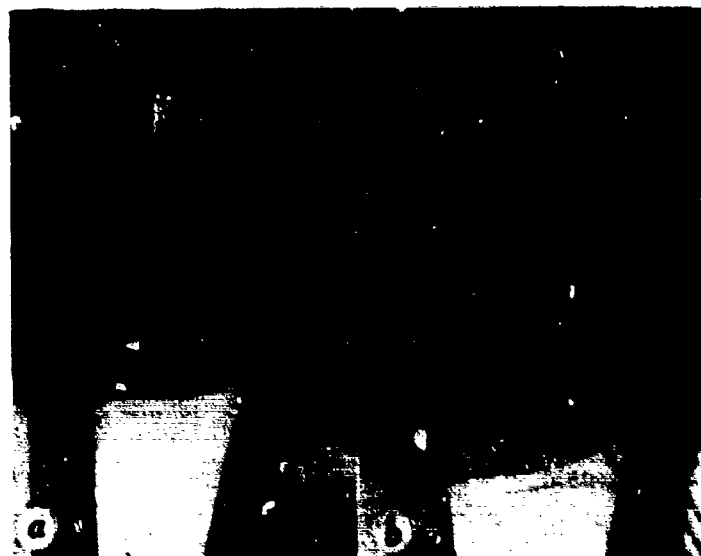
Figure 14. Subject on the human centrifuge. (a) At 1 G prior to centrifuga-
tion. (b) At 5 G during rotation of centrifuge. Note because of
increased weight, the appearance of increased age. The mouthpiece
contains a thermocouple for recording respirations (as temperature
variations) in the airway. The two photoelectric earpieces sup-
ported on the headband are used for recording the blood content of
the ear (ear opacity) and the change in opacity of the ear pro-
duced by each cardiac systole (ear opacity pulse). The ECG was
recorded simultaneously from bilateral chest electrodes, 1943.
(Reproduced with permission from E.H. Wood, Mayo Clinic
Proceedings 1975, 50:497.)

A photokymographic recording from such a subject during a 5-sec exposure
to +4 G is illustrated in Figure 15. This recording was the standard 5-sec
acceleration exposure profile set up in 1942 by the Toronto group and adapted
initially by the group in Rochester, Minnesota on the basis: (1) that
aircraft, of that time, did not have the power to sustain accelerations at 4 G
or above for more than 5 sec, and (2) at that time, considered most important,
that circulatory collapse would result due to the presumed cessation of venous
return to the heart, which was believed to occur if high levels of positive
acceleration were sustained for more than 5 sec.

Figure 15. Photokymographic recordings from an experienced, relaxed,
30-year-old male centrifuge subject during a 5-sec exposure to
a plateau level acceleration of 5 G in the upright sitting posi-
tion. Vertical white lines delineate 5-sec intervals. From
top downwards: EP, ear opacity pulse; ECG, electrocar-
diogram; R, respiration; EO, ear opacity; G, acceleration
in G units; LR, responses to light signals at the visual fixation
point; BR, responses to buzzer signals. Note the progressive
decrease in ear opacity (blood content) of the ear throughout the
exposure and that the ear opacity pulse was obliterated
(indicating zero systolic arterial pressure at head level [54])
throughout the 5-sec exposure to 5 G. The subject reported
transient dimming of vision at the end of exposure (1942).

This hypothesis was tested in late 1942, with the, at that time,
surprising results illustrated by recordings (Figure 16) of changes in heart
rate and circulation to the ear as the duration of exposures to the same level
of acceleration was increased from 5 up to 30 sec.

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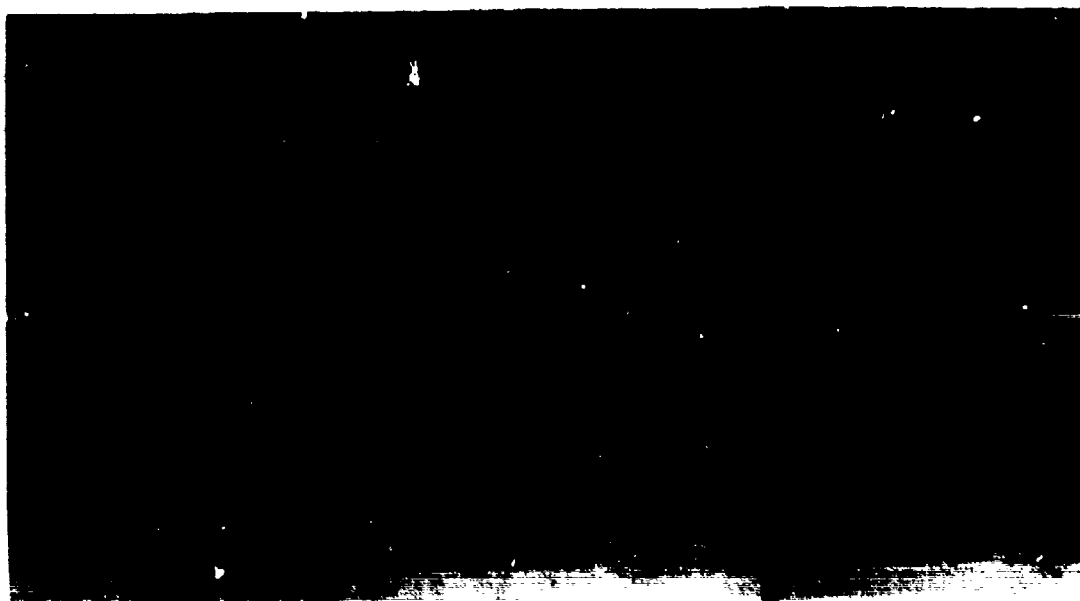
Figure 16. Changes in circulation to the ear and associated visual symptoms with the duration of exposures of a healthy male subject to plateau acceleration levels of 4 G when in the upright sitting position. Right panel: A transient dimming of vision occurred at the termination of the 5-sec exposure. Center panel: Peripheral vision was lost 6 sec after attainment of the 4 G plateau and, following recovery of the ear pulse and blood content of the ear, was maintained clear from the 12th throughout the balance of the 30-sec exposure. Left panel: This is an example of the "standard" 15-sec exposure to plateau levels of acceleration attained at a rate of about 2 G/second above 1.5 G. This duration of exposure covers the initial about 7-10 sec of progressive failure of circulation to the head, the resulting losses of vision and/or consciousness and the reflex cardiovascular compensatory changes which occur in relaxed healthy humans (56). This acceleration profile was used for the majority of assays of G tolerance and the protective value of various protective devices and maneuvers from 1942 through 1946 on the Mayo Human Centrifuge (43) and in a specially instrumented dive bomber (40). See legend of Figure 15 for additional details.

The initial period of progressive failure was terminated at about 10 sec by surprisingly effective compensatory changes associated with return of arterial pulsations and blood content of the ears, a decrease in heart rate, and actual recovery of vision sustained throughout the last 20 sec of the 30-sec exposure.

This unexpected finding was dramatic proof that much more than simple hydrostatic predictions were involved in the G-induced losses of vision and consciousness -- in other words, that a great deal of exciting acceleration physiology must be involved. Various aspects of force environment physiology have

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been under investigation since that time and in spite of 4 decades of continued research are still not fully understood (57).

Measurements of systemic arterial pressure were of primary importance for elucidating the major circulatory effects of sustained exposures to headward acceleration. Adaptation of the strain gauge principle for measurement of blood pressure was developed at Mayo for this purpose (58). Figure 17 is a picture taken in the cockpit of the human centrifuge of Dr. Ed Lambert, who played a major role in these studies, with an early model strain gauge manometer coupled via malleable lead tubing to an indwelling 19-ga needle in his left radial artery rigidly supported at base of brain level for recording during exposures to acceleration (59). Simultaneous, direct, continuous recordings of arterial pressures from both radial arteries (Figure 18)--one supported at base of brain and the other at base of heart level--were most revealing.
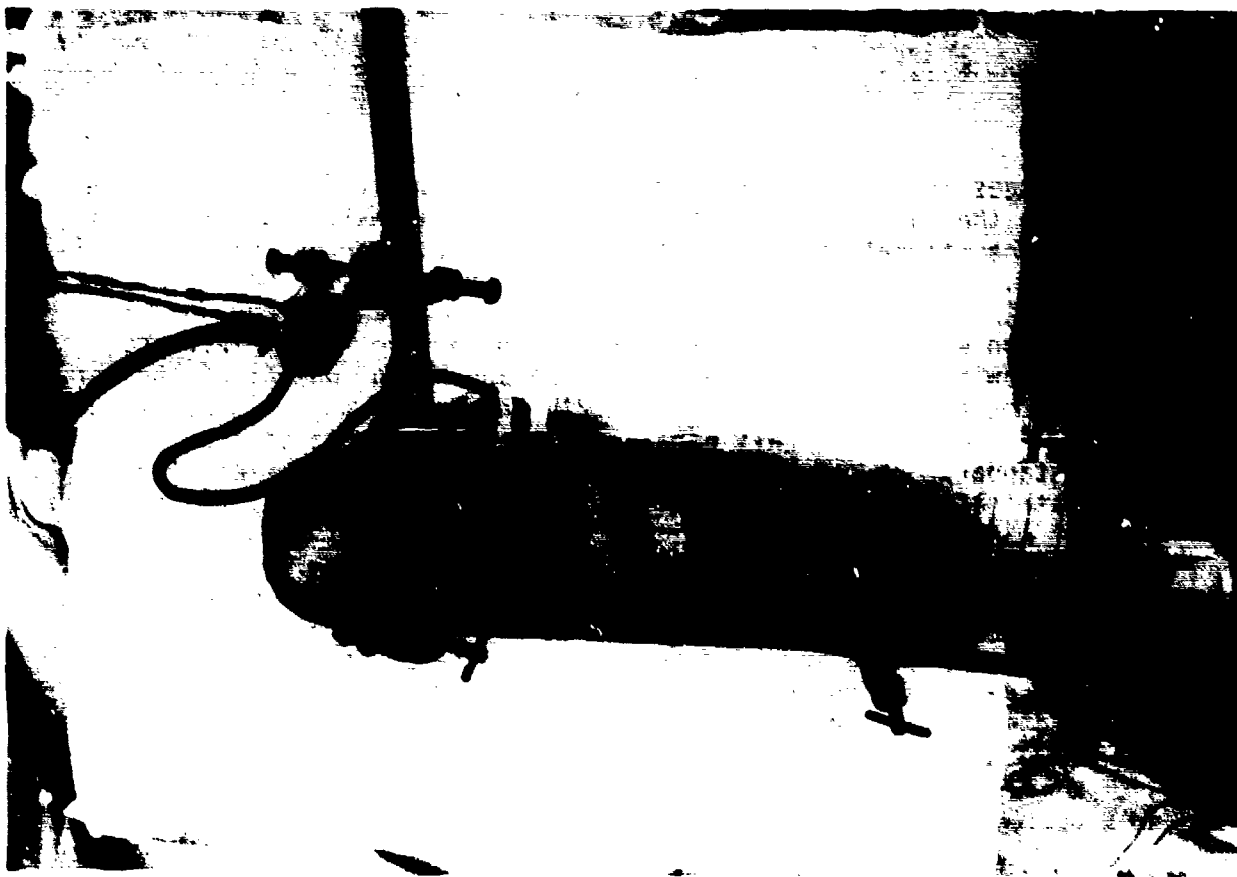
Figure 17. Assembly used for continuous direct recording of arterial pressure in the radial artery on the Mayo Human Centrifuge. The position of the recording site was maintained at the desired level relative to the heart and/or brain during acceleration by means of the rigidly mounted arm board and wrist support (58). (Reproduced with permission from E.H. Wood et al., Indwelling and Implantable Pressure Transducers. Cleveland: CRC Press, 1977, pp. 21-34.)
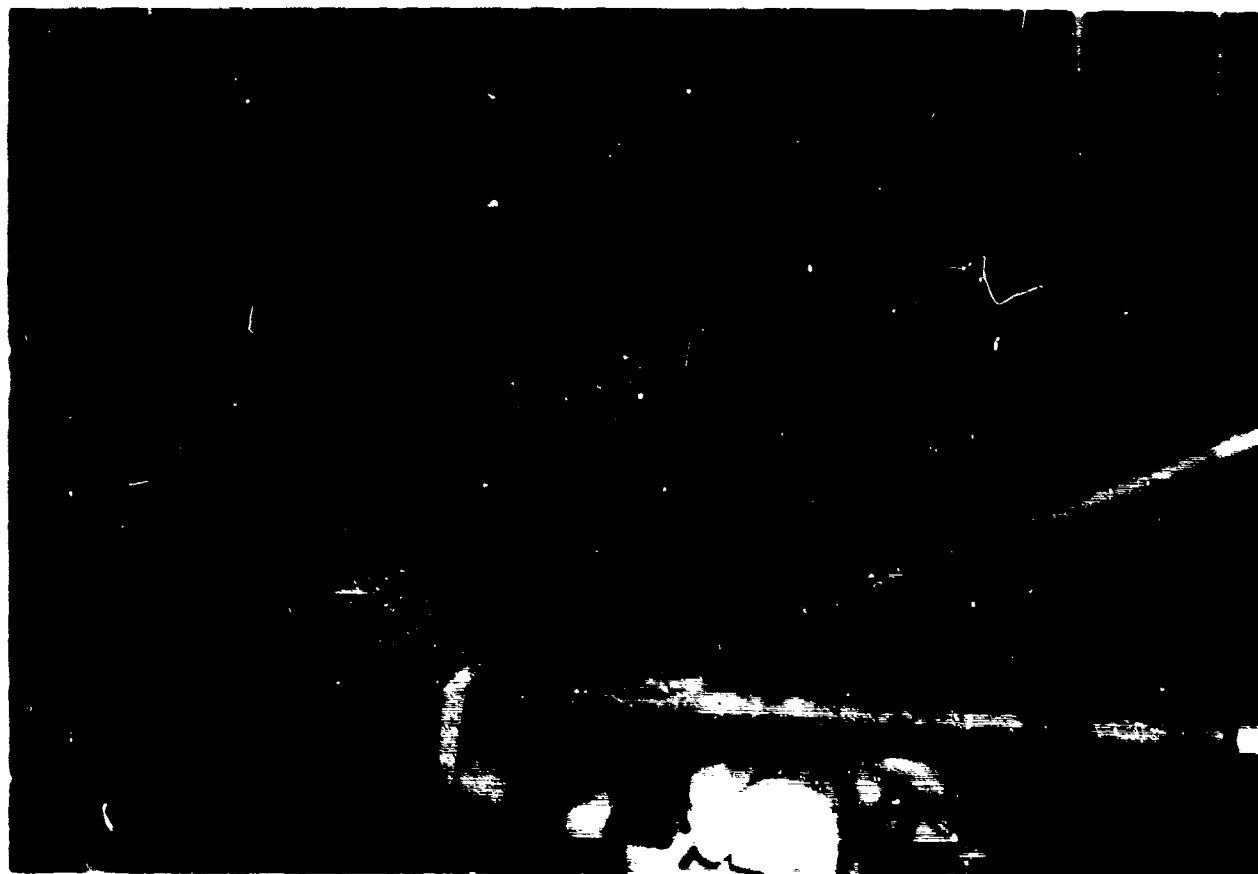
Figure 18. Position of subject with indwelling needles in both radial
arteries (Figure 17) for simultaneous recording of systemic
arterial pressures at heart and head levels during exposures to G_z
acceleration on the Mayo Human Centrifuge. The subject is wearing
a plaster helmet which provides a firm support for the pno-
toelectric earpiece shown on the right ear. Figure 19 is an
illustration of multiparameter recordings obtained with this assembly.

Figure 19 is a 1945 photokymographic recording of 12 simultaneous phy-
siologic parameters during a 15-sec exposure of a healthy young man to an
acceleration of 4.5 G. Note particularly, the arterial pressure was maintained
normal at neart leve᠊ in spite of the expected decrease in venous return from
the dependent portions of the body, and that after the initial 7 sec of the
exposure a dramatic increase in arterial pressure occurred, which was suffi-
cient to overcome the 4.5-fold increase in hydrostatic distance up to the
brain so that vision was restored during the last few seconds of this 15-sec
exposure.
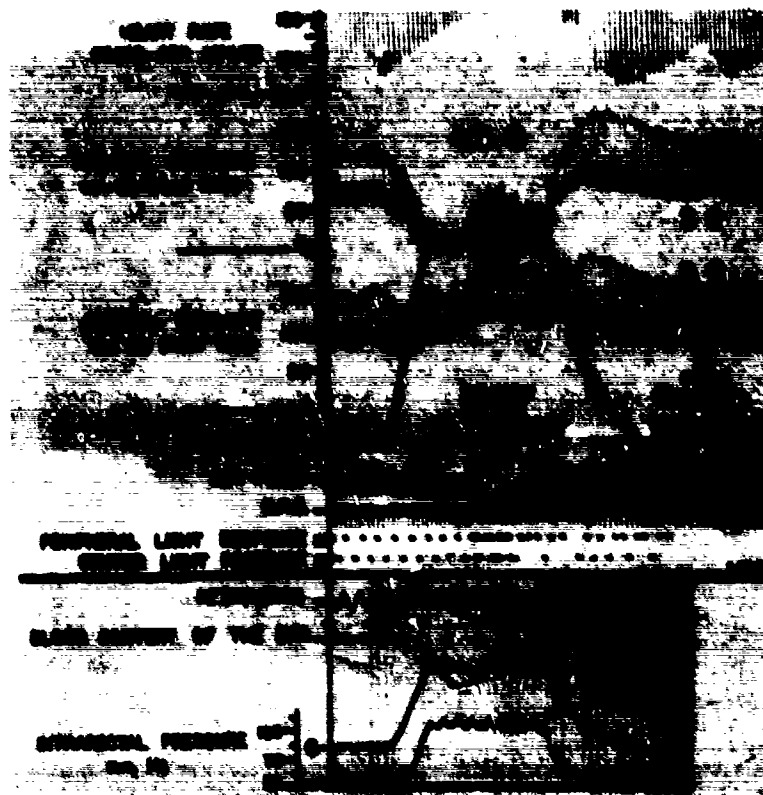
Figure 19. These recordings were obtained by two photokymographic cameras
operating simultaneously, one mounted in the centrifuge cockpit
(lower panel) and one in the recording room adjacent to the
centrifuge (upper panel). Vertical white lines on the upper panel
delineate 5-sec intervals and were 15 mm apart before photographic
reduction. Black acceleration line indicates the magnitude of
headward acceleration in G units. Simultaneous recording of
acceleration (indicated as G in lower panel) serves to synchronize
the two recordings. Length of black lines, designated as
peripheral and center light response, indicates subject's reaction
times to light signals in peripheral and central fields of vision,
respectively (see text for further details).

How is this possible? A major reason is indicated by the bottom tracing
of intrarectal pressure which is directly related to intraperitoneal pressure
in the dependent regions of the abdominal cavity.

At 1 G the intraabdominal pressure of about 25 mmHg was sufficient to
maintain a column of venous blood up to the dome of the diaphragm. This
pressure increased in direct proportion to the force environment so that at
4.5 G it was about 125 mmHg and varied with the vertical height of the
diaphragm during each respiratory cycle.

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In other words, as first postulated by Dr. Robert Rushmer,(60), and later by Duomarco and Rimini (61), the abdominal contents behave like a hydrostatic system, that is, as if the abdominal cavity were filled with water. Therefore, venous return from the abdomen to the heart is maintained during headward acceleration so that systemic arterial pressure is well sustained and a compensatory hypertension can occur in the attempt to maintain cerebral circulation.

This system is one of nature's very effective internal G-suits (62). Figure 20 is a plot of average intrarectal pressure values in nine healthy men in the force environment range from 1 to 5 G. The solid straight line is a linear extrapolation from the 1 G value. The fall off from this line is due to the progressive descent of the diaphragm caused by the increase in weight of the abdominal and thoracic contents with increases in G level.

EFFECT OF POSITIVE ACCELERATION ON INTRARECTAL PRESSURE
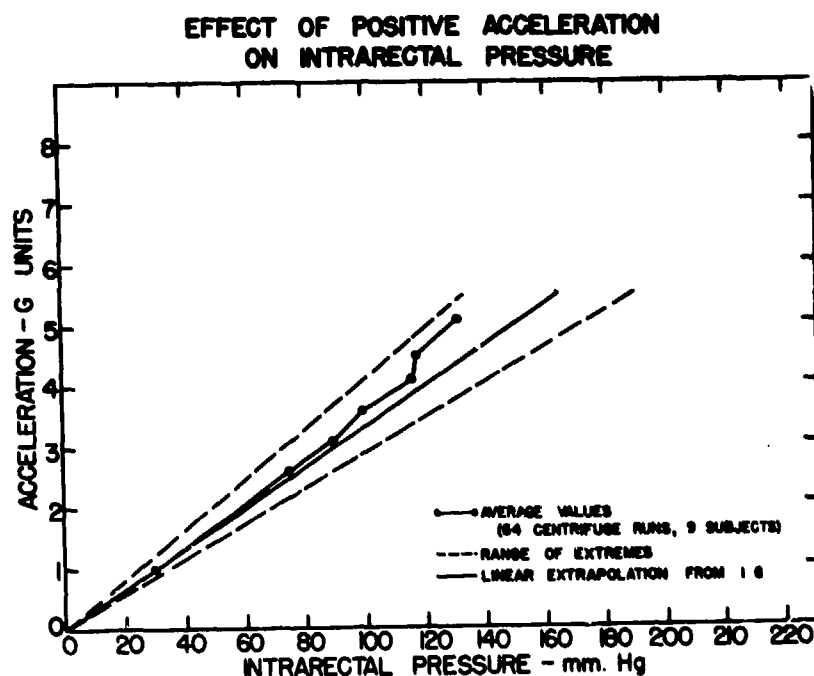
Figure 20.   Effect of G_z acceleration on intrarectal (intraabdominal) pressure in nine relaxed male subjects in the upright (13° from the vertical) sitting position.

As illustrated in Figure 21, inflation of a G-suit reverses this fall of intraabdominal pressure thus creating a venous pressure gradient from the abdominal organs back to the heart.

EFFECT OF POSITIVE ACCELERATION ON INTRARECTAL
PRESSURE WITH AND WITHOUT SUIT PROTECTION
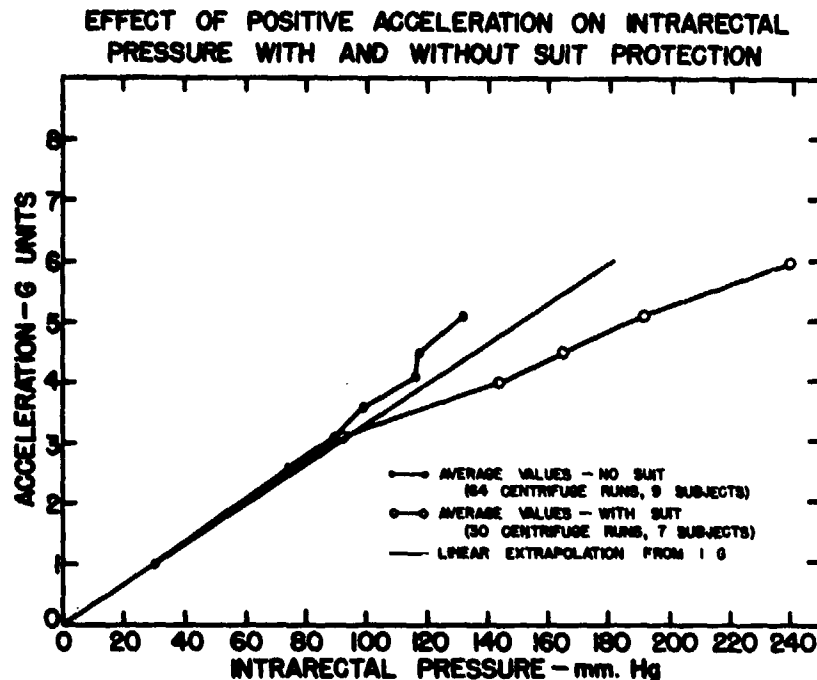
Figure 21. The effect of headward acceleration on intrarectal (intra-
abdominal) pressure with and without inflation of a pneumatic
anti-blackout suit. The straight line is the intrarectal pressure
that would be predicted if the abdominal contents acted as a per-
fect hydrostatic system of fixed height during acceleration. Note
(1) that the pressures recorded without the protection of the suit
fall slightly but progressively below this line at the higher
levels of acceleration. This result is related to the reduction in
height of the hydrostatic column due to the descent of the
diaphragm and the bulging outward of the abdominal wall caused by
the acceleration; and (2) that the inflation of the anti-blackout
suit and the consequent application of external pressure to the
legs and, particularly, to the abdomen (see Figures 10-12) was
associated with increases in intraabdominal pressure to values
considerably above those required to support a column of blood
from the dependent portions of the abdomen up to the base of the
heart. The pressure in the bladder system of this suit was
controlled by a valve automatically activated and compensated
according to the acceleration; the suit pressure, which was zero
at 1.5 G, increased in proportion to the acceleration at the rate
of 52 mmHg per G above this level. At an acceleration of 6 G
there was approximately a one-to-one relationship between the
intrarectal pressure and the pressure to which the bladder system
of the suit was inflated. See Figures 19, 22, 24-29 for examples
of recordings of intrarectal pressure from which the plotted
values were obtained. (Collection and analysis of these data were
carried out by Lambert and Wood, 1946.)

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The resulting increase in preload to the heart simultaneously with an increase in afterload are the major causes of the increase in blood pressure and consequent increases in G tolerance produced by inflation of a G-suit as illustrated in Figure 22.

Figure 22. Objective recordings which document the protection against the circulatory and associated effects of headward acceleration afforded by inflation of an anti-blackout suit. During the control exposure of this normal subject to 4.6 G_z (left panels), arterial pressure was reduced to zero at head level, and complete loss of vision resulted. The effects of inflating the bladder system of the suit to approximately 225 mmHg at 1 G and to the same pressure during an exposure to 5.5 G are illustrated in the center and right panels, respectively. Pronounced bradycardia and other depressor reflexes limit the development of the hypertensive effect produced by inflation of the suit at 1 G. During the exposure to 5.5 G, arterial pressure at heart level was increased to 200/140 mmHg or more; perfusion of the head was, therefore, maintained to the degree that only dimming of vision occurred at this level of acceleration, which was approximately 1 G greater than the level of acceleration producing complete loss of vision without the suit. Note (a) the correlation between the intrarectal (intraabdominal) and suit pressures (lower panels); at this level of acceleration the intraabdominal pressure was only slightly less than the pressure in tne bladder system of the suit, and (b) the decrease in amplitude of respiration at 5.5 G caused by the high intraabdominal pressure that elevates and restricts movement of the diaphragm. (See Figure 19 for detailed explanation of tracings) (with permission [19].)

Self-induced transient increases in blood pressure at heart level are also the basis of voluntary muscular-respiratory self-protective straining maneuvers such as the M-1, the L-1,and pressure breathing, all of which are variants of the Valsalva maneuver (18).

The well-known, to most of you, changes in blood pressure and heart rate produced by a voluntary increase in airway pressure of 60 mmHg for 15 sec (i.e., a classical Valsalva maneuver) are illustrated in Figure 23. The increase in arterial pressure at the onset of the expiratory effort is respon- sible for its anti-G effects. The secondary decrease in arterial pressure and consequent decrease in G tolerance, if the expiratory pressure is held for longer than 5 sec, is responsible for the two-edged sword characteristics of this maneuver.
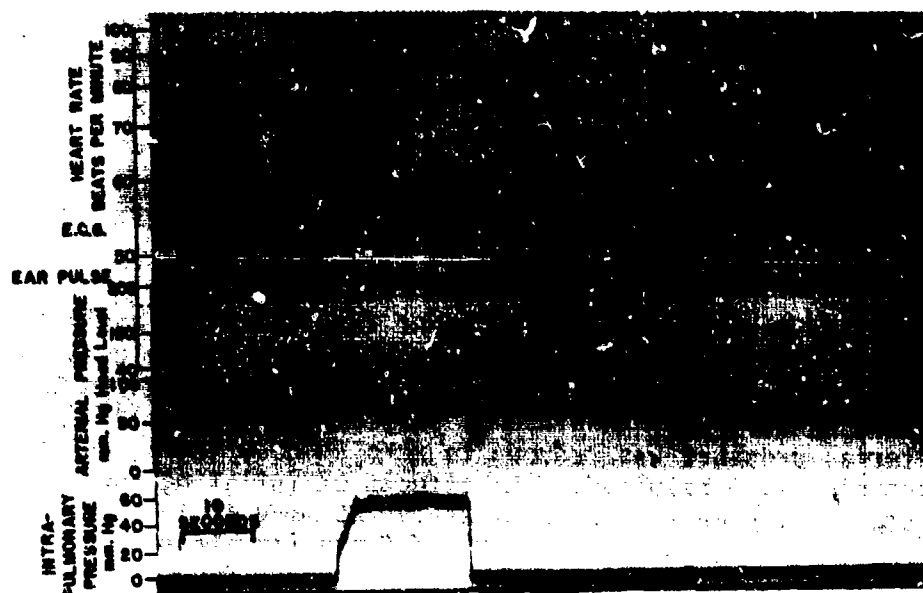
Figure 23.   Arterial pressure and heart rate effects of a 15-sec sustained 60 mmHg increase in airway pressure in a healthy male subject sitting upright.

The secondary, potentially dangerous decrease in arterial pressure can be avoided by slowly exhaling through partially closed lips or glottis during each period of expiratory effort so that a quick inhalation is required every 3-5 sec in order to maintain the airway pressures. This mode of action of the so-called M-1 manuever is illustrated in Figure 24.

24
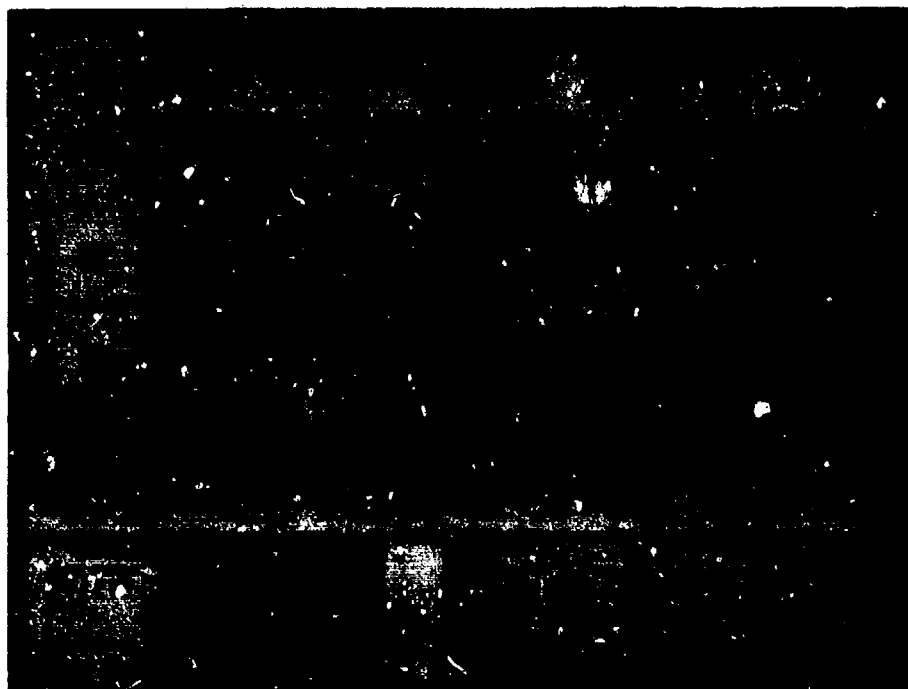
Figure 24. Recordings illustrating the protection against blackout by the M-1
           maneuver. In this experiment, the successive 3-5 sec Valsalva
           maneuvers were performed with the glottis open and the lips closed
           over an oral airway connected to a controlled leak strain gauge
           manometer system. In the control exposure of this healthy male
           subject to 5 G (left panel), arterial pressure was reduced to zero
           at head level, and a complete loss of vision resulted. The center
           and right panels show the effects of the M-1 maneuver when per-
           formed with the centrifuge stationary, that is, at 1 G, and during
           an exposure to 5 G, respectively. The hypertension produced at
           heart level was sufficient to maintain perfusion of the head so
           that vision was preserved throughout the exposure to 5 G. Note
           that the effects of the M-1 maneuver and acceleration on the
           intrarectal pressure are additive, so that intraabdominal pressure
           increased to more than 200 mmHg during the exposure to 5 G; this
           is far in excess of the approximately 60 mmHg generated in the
           pulmonary airways during the cycles of expiratory straining. This
           pressure gradient from the abdomen to the thorax is greatly
           accentuated during each inspiration and apparently acts to pump
           venous blood back to the heart during this phase of the forced
           expiratory cycle. (°ee Figure 19 for detailed explanation of
           tracings.)

    The action of the L-1 maneuver is similar, as illustrated in the left
panel of Figure 25, but with the important difference that the expiratory
pressure is maintained with a closed glottis. This difference necessitates
both expiration and inspiration during the breath between each expiratory

effort and so approximately doubles the duration of the undesirable period of decreased blood pressure which accompanies each breath as compared to the M-1 illustrated in the right panel. The so-called L-1 is somewhat easier to do and does provide a similar degree of protection (Figure 26).
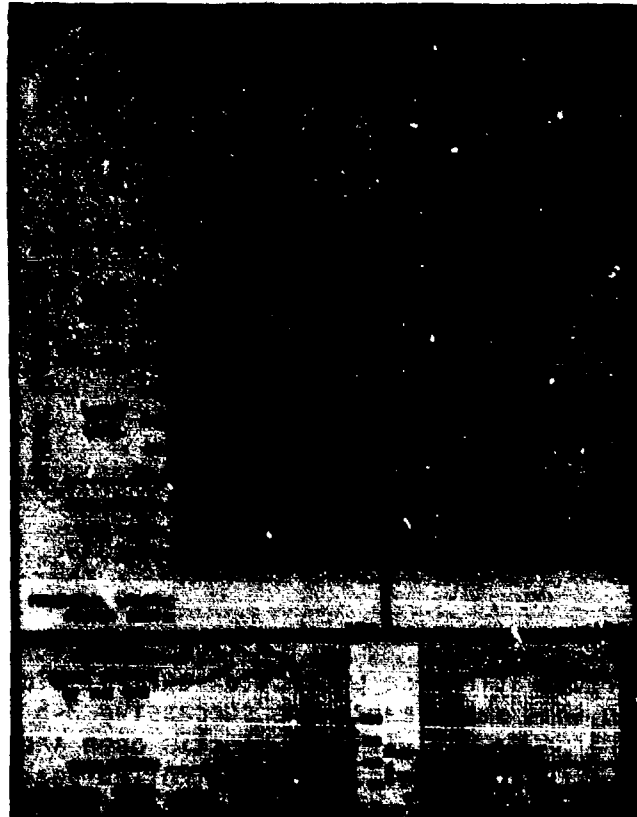
Figure 25.   Recordings illustrating the effects of successive approximately 3 to 5 sec duration Valsalva maneuvers monitored by (1) a closed manometer system (left panel) and (2) a slowly leaking system (right panel). When a closed manometer system is used, both expiration and inhalation are required between each successive increase in intrapulmonic pressure (left panel). The 2-3 sec duration of the decreased arterial pressure associated with each expiratory-inspiratory cycle is more than double the approximately 1-sec period required for inspiration only, which is the case when a slowly leaking manometer system is used (right panel). Note the third period of expiratory effort in the left panel: This period of increased airway pressure was maintained for more than the optimum 3-5 sec, as can easily occur when a closed system is used. Consequently, the phase 2 decrease in arterial pressure of the Valsalva maneuver began, which, if maintained during an exposure to acceleration, could result in GLOC at surprisingly low levels of acceleration. This possibility is avoided when a leaky manometer system is used because the progressively emptying chest, during each period of expiratory effort, mandates an inhalation every 3-5 sec in order to maintain the airway pressure (right panel).
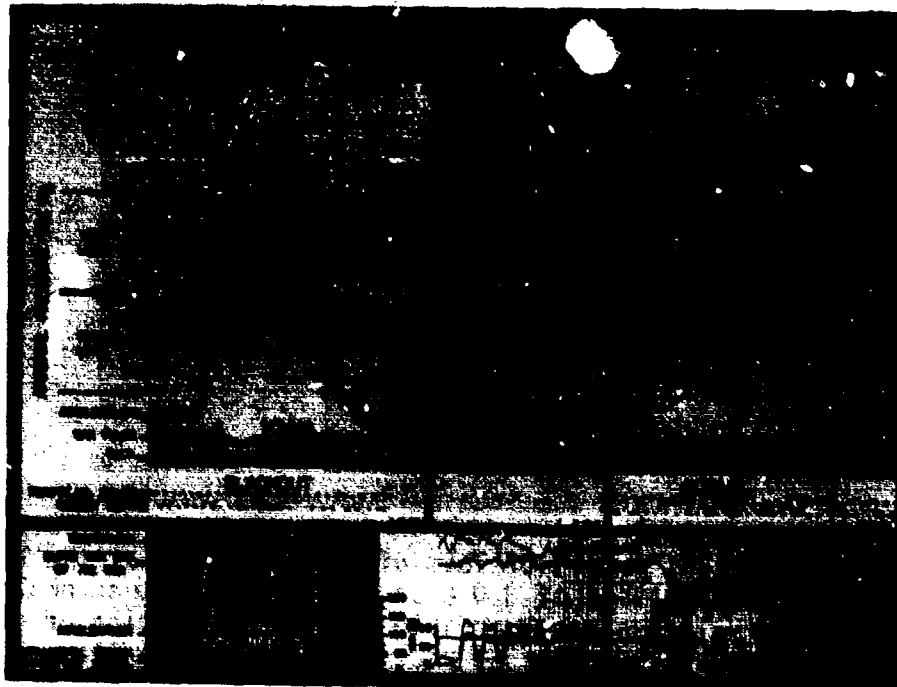
Figure 26. Recordings illustrating the protection against blackout by use of successive brief Valsalva maneuvers performed with the glottis open and lips closed (the L-1 maneuver). Intrapulmonic pressure was monitored via a catheter held between closed lips using an air-tight strain gauge manometer. The recordings in Figures 24-26 were obtained from the same subject. (See legend of Figure 24 for more details.)

However, because the glottis is kept closed during the expiratory effort, the L-1 maneuver enhances the chances of inadvertently prolonging the period of expiratory effort into the secondary, potentially dangerous decrease in arterial pressure phase of the classic Valsalva maneuver (Figure 23).

If this maneuver is done during an exposure to acceleration, GLOC can occur at unexpectedly low levels of acceleration, as illustrated in Figure 27 which documents an incident of GLOC at the surprisingly low level of 3 G precipitated by a prolonged Valsalva maneuver.
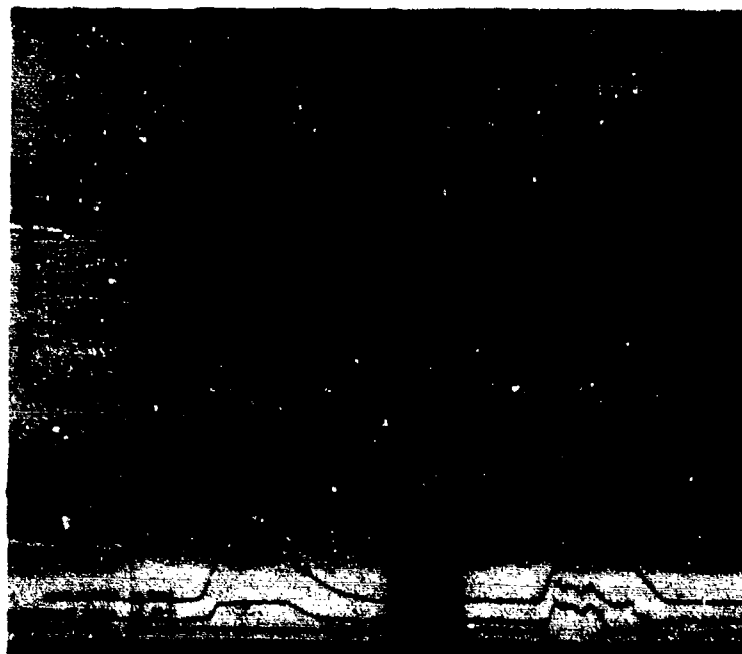
Figure 27. An example (right panel) of loss of consciousness at the surprisingly low acceleration of 3.0 G caused by inadvertent prolongation of the Valsalva maneuver into the phase 2 period of decreased arterial pressure. Note that recovery of responses to peripheral and central light signals did not occur until 18 sec after the return to 1 G. This situation is much more likely to occur when the glottis is closed during each 3-5 sec period of expiratory effort as used in the L-1 type self-protective maneuver.

It is of interest that although the M-1 maneuver was demonstrated during World War II to provide about 2 G more protection than the standard U.S. Air Force G-suit, it was considered to be a laboratory curiosity which was too distracting and physically fatiguing for practical operational use. However, with the advent of very high speed, maneuverable jet-powered aircraft, it soon became evident that the protection afforded by the G-suit alone was inadequate.

The fortunate fact discovered in 1943 that the protection afforded by a G-suit and voluntary straining maneuver are additive (19) was the basis upon which the late Dr. Sidney Leverett and colleagues at Brooks Air Force Base, about 20 years later, were able in a series of multifaceted pioneering studies, to demonstrate the amazing fact that well-trained centrifuge subjects and many fighter pilots could, by combining the M-1 maneuver and the G-suit, maintain clear vision at 9 G sustained for 45 sec and longer. These heroic studies constitute landmarks in acceleration physiology which merit the gratitude and admiration of all workers in this field (15,16).

However, in spite of this capability, incidents of crashes due to GLOC, as indicated in Figure 3, have occurred at alarming frequency during the last several years (14,63). This situation has been dramatized in several magazine articles and video news segments (20-22).

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The dramatic and quite certainly life-saving effect of inflation of a G-suit in reversing the decrease in blood pressure which otherwise would occur if a pilot doing the L-1 maneuver inadvertently performs a sustained Valsalva, is illustrated in the first and third panels of Figure 28 of continuous arterial pressures at heart and head levels along with ten other simultaneous variables, of which the airway and intrarectal pressures are, from the mechanistic viewpoint, most important. Note that the secondary phase 2 decrease in arterial pressure due to the Valsalva maneuver at 1 G is much more severe and hence potentially dangerous during an exposure to 3.5 G (second panel). However, the dramatic increase in intrarectal pressure which occurs when the G-suit is inflated both at 1 G (third panel) and during an exposure to 6.5 G (fourth panel), provides a pressure gradient from the abdomen to the thorax which maintains venous return from the abdomen. Consequently, the secondary decrease in arterial pressure is abolished and a sustained hypertension results.

Figure 28. Objective recordings which document (1st and 2nd panels) that G_z acceleration exacerbates the deleterious circulatory effects of a continuous Valsalva maneuver and that inflation of an effective anti-G suit (3rd and 4th panels) not only abolishes this deleterious effect but, because of the combined effect on intra-abdominal and intrathoracic pressures (lower right panels) and the resulting decreased compliance of abdominal and pulmonary vasculature, actually enhances the hypertension produced by inflation of the suit.

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Furthermore, as shown in Figure 29, performance of a Valsalva maneuver concomitantly with inflation of a G-suit increases the level of hypertension produced by suit inflation at 1 G (first panel) and also during an exposure to 6.5 G with a corresponding increase in G protection. With the suit alone, a complete loss of vision occurred at 6.5 G (second panel) whereas vision was only dimmed at the same acceleration with combined inflation of the suit and a Valsalva (right panel). Note particularly that with the Valsalva and suit inflation the intrarectal pressure of nearly 250 mmHg at 6.5 G approached the suit pressure of about 300 mmHg and exceeded intrapulmonic pressure by about 150 mmHg, thus creating a large pressure gradient from the abdomen to the thorax and assuring venous return to the heart.
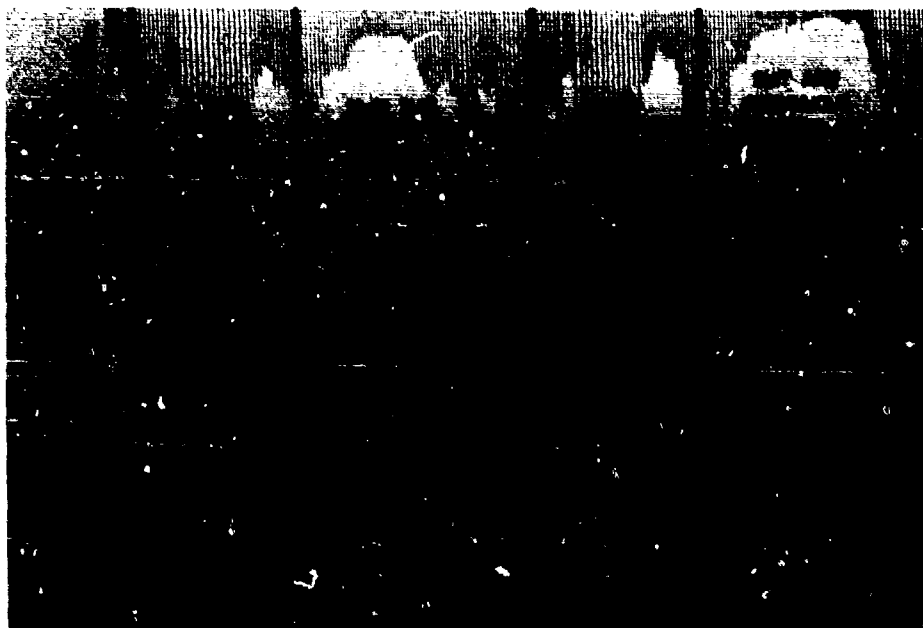
Figure 29. Effect of performance of a continuous Valsalva maneuver on the cardiovascular reactions produced in the same subject by inflation of an anti-blackout suit to the pressure used for 6.5 G of positive acceleration when the subject was seated at 1 G, and on the protection afforded by inflation of the suit during exposure to positive acceleration of 6.5 G.

The importance of intraabdominal pressure as a determinant of the effect of a Valsalva maneuver on arterial pressure is dramatically illustrated in Figure 30, obtained in collaboration with Dr. Hilding Bjurstedt (Head of the Human Centrifuge at the Karolinska Institute in Stockholm) back in 1953 (64). When the increases in intrapulmonic and intraabdominal pressures were equal, (left panel) so that the abdominal to thoracic pressure gradient was zero, the

impediment to venous return from the periphery resulted in a decreased
arterial pressure from about 150 to 100 mmHg during a 10-sec Valsalva
maneuver. When the same increase in airway pressure was produced (right
panel) without a concomitant increase in intraperitoneal pressure to support
venous return from the abdomen, a much more severe decrease in arterial
pressure resulted.

Figure 30. Effects on the arterial pressure and heart rate during raised air-
            way pressure. Dog, Nembutal anesthesia. (A) Effects of
            increasing both the intrapulmonic and the intraperitoneal pressure
            to the same level (40 cm H₂O). (B) Effects of increasing only the
            intrapulmonic pressure (40 cm H₂O). Time marking: 1 min between
            vertical lines. (From Bjurstedt and Wood [64]).

The opposite effect can be produced by increasing intraabdominal pressure
to higher levels than intrapulmonic. This effect is a major basis of the M-1
maneuver (Figures 24 and 25) and the dramatic additional protection which
occurs, when the increases in intraabdominal pressure produced by the Valsalva
maneuver and the G-suit are additive (Figure 29).

The intensity of effort required to maintain clear vision at 9 G by use of
the M-1 maneuver and inflation of a G-suit, such as is routine procedure for
modern fighter pilots, can be best appreciated by viewing colored cinefilms
(38) recorded during sustained exposures to 9 G with clear vision, achieved by
combined use of a very effective progressive arterial occlusion suit (Figure
31) and the M-1 maneuver. Figures 32 and 33 of objective recordings obtained
during this February 1943 exposure to 9 G are of considerable interest.

31
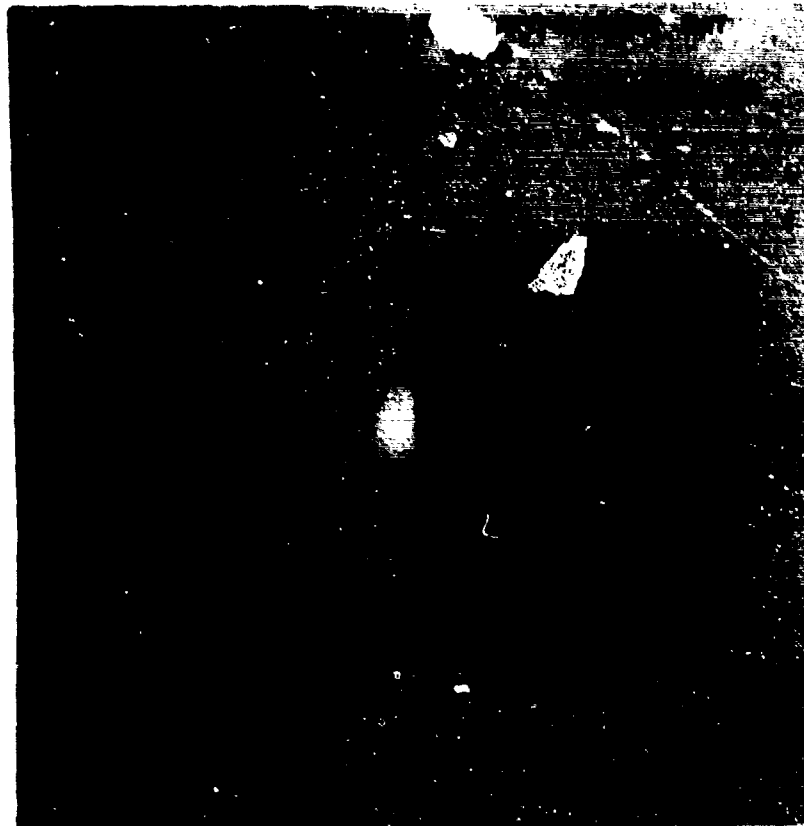
Figure 31. Progressive arterial occlusion anti-G suit fabricated by David M. Clark Mayo Human Centrifuge Laboratory, 1942. Arterial occlusion suits were found to provide much higher levels of protection than devices designed to support venous return. This model with its: 60 lb/square inch pressure source (1), and very rapidly inflating pressure control valve (2) was the most effective; (3) pressure inlet line to bladder system - arrows indicate direction of air flow cephalad from both ankles during the inflation phase of suit pressurization which is initiated automatically at accelerations greater than 1.5 G_z; (4 and 5) segmented calf and thigh bladders, respectively; (6) segmented abdominal bladder; (7) outlet bladder pressure control line via which the valve automatically adjusts the final bladder pressure to the desired values and vents the suit at accelerations of less than 1.5 G_z; (8) internal and external zippers which unite the right and left segments of the abdominal bladder into a single system (lower panel); and (9) arterial occlusive arm cuffs. The very rapidly progressive high-pressure inflation of the bladder system from the ankles to the abdomen was controlled by the pressure shut-off line from the top of the abdominal bladder (7), which regulated the final level of pressure according to the level of acceleration. This pressure was set to the presumed arterial occlusive level of 150 mmHg at 1.5 G and increased automatically by 50 mmHg per G above this level - so, for example, at 5 G the final suit pressure was about 325 mm Hg.

Figure 32 shows a series of strip-chart recordings; from above downwards: the reaction times to peripheral light signals, the ear opacity pulse, reaction time to buzzer signals, and the electrocardiogram.

32
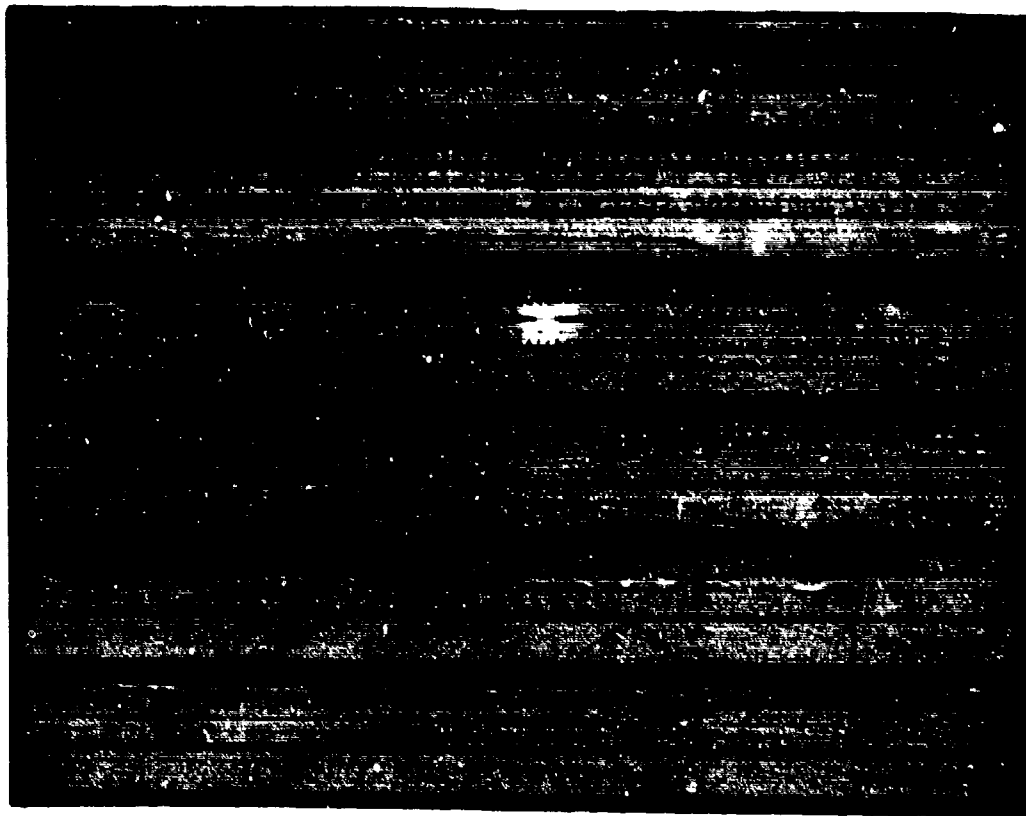
Figure 32. Objective, 6 channel, oscillographic recordings obtained February 16, 1943 used for measurement of increase in G tolerance of a healthy male human centrifuge subject provided by a progressive arterial occlusion suit (Figure 31) and the additive protective value of the suit and the M-1 self-protective maneuver. Note that in the control exposure (i.e., suit not inflated), the ear pulse was obliterated (indicative of zero systolic pressure at head level [54]) during the initial 7 sec of the 30-sec plateau exposure to 5 G during which, concomitantly with failure to respond to peripheral light signals (top tracing) from the 4th to 15th sec of the plateau G level, the subject reported complete loss of vision followed, subsequent to recovery of the ear pulse, by clearing of vision and secondary dimming and loss of peripheral light responses toward the end of the 30-sec exposure associated with secondary decreases in amplitude of the ear opacity pulse. The ear pulse was maintained during 6-G and 8-G exposures with the suit inflated, but a transient loss of peripheral light responses occurred at 8 G. The ear pulse and clear vision were maintained throughout the exposure to 9 G with simultaneous inflation of the suit and the M-1 maneuver.

The top three strips are control relaxed runs (i.e., with no pressure in the suit) at 3, 4, and 5 G. The ear pulse was obliterated and a blackout resulted at 5 G.

With pressure in the suit, the ear opacity pulse was maintained at 5 G and 8 G, and also at 9 G, when with the use of the suit plus the M-1 maneuver, vision remained clear throughout the exposure.

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Simultaneous photokymographic recordings of respiration and the ear opacity from the same exposures are shown in Figure 33. An upward deflection of the thick white trace indicates a decrease in blood content of the ear. The similar deflections during the 5-G control and the 7-G and 8-G suit runs indicate that the objectively measured protection (43) afforded by this progressive arterial occlusion suit with arm cuffs was about 2.5 G.

Figure 33.
Photokymographic recordings of the respiration (top tracing) and ear opacity (blood content of the ear, thick white trace) at increasing plateau acceleration levels. These recordings and the simultaneous oscillographic tracings in Figure 32 provide the basis for objective assay of the protection afforded by a progressive arterial occlusion suit and documentation of the additional protection provided by simultaneous use of the suit and the M-1 self-protective maneuver. The subject's report of complete loss of vision during the 5-G control exposure as compared to the reported clear vision during the 9-G run indicates that use of the suit plus the M-1 provided a protection of more than 4 G against visual symptoms. The facts: (1) that the ear pulse was lost during the 5-G control run but maintained at 9 G with the use of the suit plus the M-1, and (2) that during these same two exposures the decrease in blood content of the ear was much greater during the 5-G control than when protected at 9 G provides objective confirmation that the suit plus the M-1 provides protection of more than 4 G against the decrease in circulation at head level. The similarity between magnitude of the protection against the subjective, ischemic anoxic, visual disturbances and the objectively recorded decreases in blood flow to the head which are the causes of the visual symptoms indicates that the changes in ear circulation can be used to measure G tolerance and to provide objective measurements of the increase in tolerance produced by various devices and procedures (43,50-54).

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Of particular note is the amazing fact that with the suit and the M-1 maneuver (Figure 33, right-most lower panel), the blood content of the ear at 9 G was actually increased to more than the 1-G value for much of the exposure.

Figure 34 shows the high degree of vascular congestion in the neck extending up to ear level. A picture of the left side of this subject's torso (Figure 35) taken after this series of exposures illustrates the petechial hemorrhages in all areas of the skin over which there was no counterpressure.
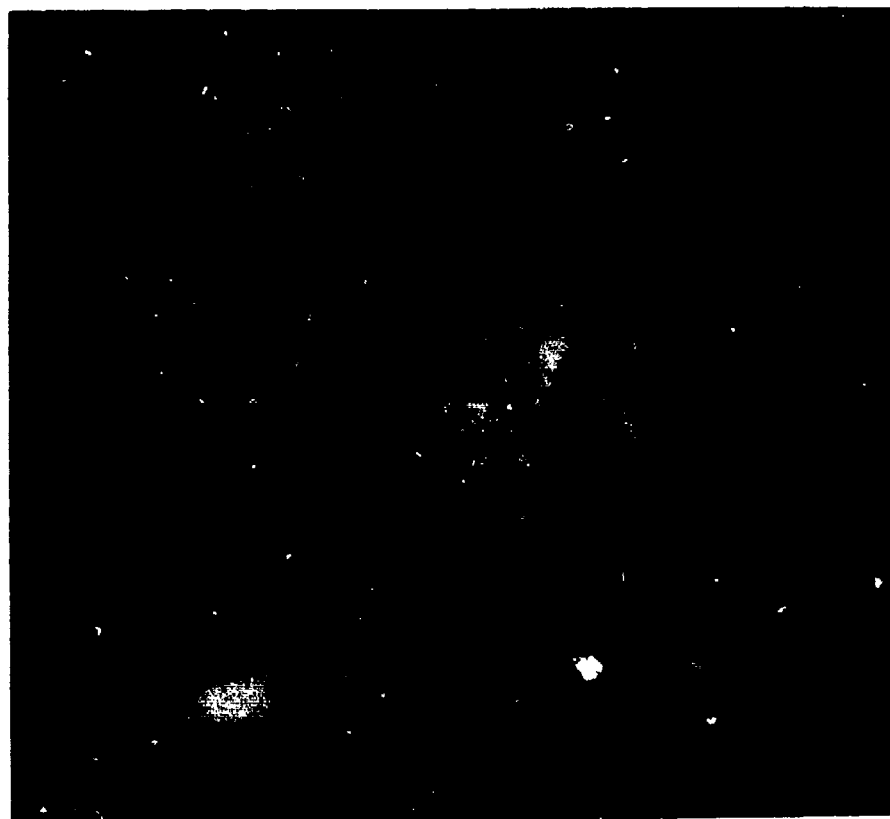
Figure 34. Single frame from a colored motion picture shows a subject during an exposure to +8 G_z protected by a progressive arterial occlusion suit (Figure 31). Note cephalad pallor which gives way to increasingly intense cutaneous vascular congestion at progressively more dependent regions of the neck and shoulders. The next to the lower right-most panel in Figure 33 includes an objective recording of the decrease in blood content of the ear which occurred during this exposure.
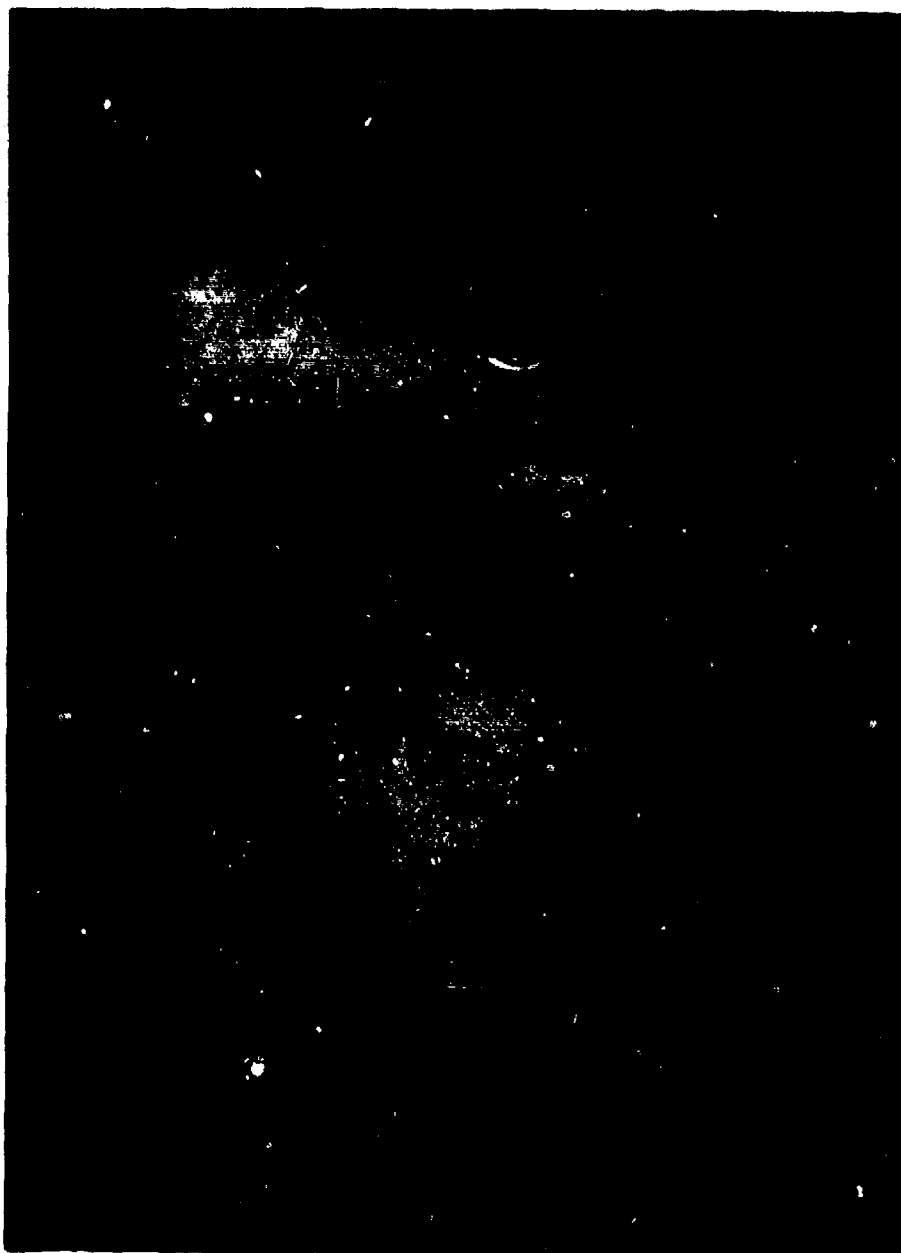
Figure 35. Petechial hemorrhages in unprotected, i.e., nonpressurized areas of the skin following maintenance of clear vision during a sustained exposure to 9 G made possible by combined use of a progressive arterial occlusion suit with bilateral arterial occlusive arm cuffs and the M-1 voluntary self-protective straining maneuver (Figures 31-34). The magnitude of the increases in intravascular pressures and associated increases in cardiac preload and after load required to produce the hypertension at aortic valve level necessary to sustain an arterial pressure at eye level sufficient to maintain clear vision during this exposure to 9 G_z were sufficient to rupture capillary endothelium in regions of the skin not protected by externally applied counterpressures. A somewhat larger subcutaneous hemorrhage is visible in the left axilla.

Note that the upper border of the arterial occlusive arm cuff on the left arm is marked by the cessation of the petechial hemorrhages visible on the left shoulder and upper arm.

These petechial hemorrhages, the axillary cutaneous hemorrhage and calculation that a systolic arterial pressure at aortic valve level of at least 300 mm Hg would be required to increase the blood content of the ear at 9 G, plus the realization that the air-filled anatomically fragile lungs and pulmonary vasculature are unprotected (66,67), convinced us that the high degree of protection afforded by a very effective G-suit and an all-out M-1 maneuver at sustained very high G levels must quite certainly be dangerous.

This belief plus the fact that such high levels of protection were not needed in fighter planes of that era resulted in the 1943 termination of studies of sustained G_z acceleration of 9 G and above on the Mayo Centrifuge.

However, the realization and foresight in the early 1970s that pilots of upcoming very high speed, jet powered, maneuverable fighter planes could be exposed to sustained acceleration in the +9-G_z range for many seconds and possibly minutes mandated resumption of the studies mentioned earlier by Dr. Leverett and colleagues at the very high sustained G levels made possible by combined use of a G-suit and voluntary straining maneuvers (15,16,68).

Their extensive series of studies have demonstrated the physiologically amazing fact and apparent safety of some trained individuals and fighter pilots to withstand accelerations in the 9-G range sustained for on the order of a minute without visual symptoms.

My own persistent fear of possible disruption of pulmonary parenchyma during high-G exposures based on physiologic considerations and reinforced by an incident on the Mayo Centrifuge of acute mediastinal emphysema in an apparently healthy subject during an exposure to 6 G (66,67) has fortunately not proved to be a problem so far.

However, the petechial and subcutaneous hemorrhages frequently seen in unpressurized areas of the skin (Figure 36) are a peripheral cutaneous indication of the anatomically destructive level of strain on the peripheral vascular system.

Figure 36. An example of
extensive petechial hemorrhages
observed after a 15-sec
exposure to +7 G_z on the
Brooks Air Force Base human
centrifuge. The subject was
using a standard USAF anti-G
suit and performing the L-1
straining maneuver. (Courtesy
of Kent K. Gillingham, M.D., Ph.D.)
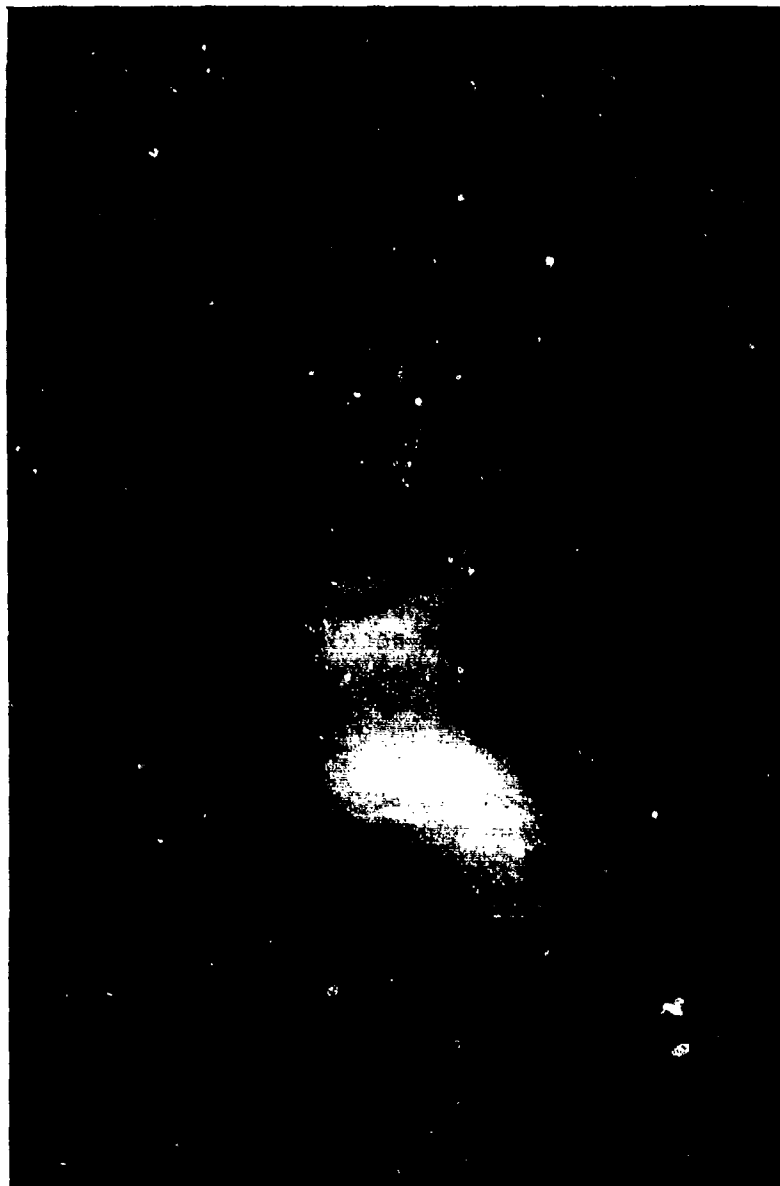
Fortunately, the potential danger of the very high arterial pressures at aortic valve level required to maintain clear vision at 9 G have as yet not materialized. However, the fact that an arterial pressure of more than 300 mm Hg at aortic valve level is required to maintain a pressure of 120 mmHg at eye level during a sustained exposure to 9 G, as has been recorded by the Brooks Air Force Base group (16), is a physiologically amazing feat, but from the cardiovascular viewpoint, somewhat frightening.

In any event, the facts suggest that, in the upright sitting position, sustained acceleration in the 9-G range should be considered the upper, reasonably safe limit of human G tolerance.

However, the multiple incidents of GLOC-related fatal crashes, as illustrated in Figure 2, indicate that the pilot's life and his plane are riding close to the brink of GLOC-induced disaster at, in some instances, levels of acceleration considerably below the 9-G range.

The apparent two most plausible causes of GLOC at relatively low G levels are: (1) an improperly performed Valsalva type straining maneuver as illustrated in Figure 27, and (2) the development of a low blood pressure in flight due to a vasovagal type reaction.

Such an incident is illustrated in Figure 37 which is based on continuous recordings of heart rate and arterial pressure at heart level in an experienced healthy volunteer centrifuge subject in what was to be a 10-min exposure to 3.5 G during which cardiac output was to be determined by the dye dilution technique at 2-min intervals before, during and after the exposure (69).
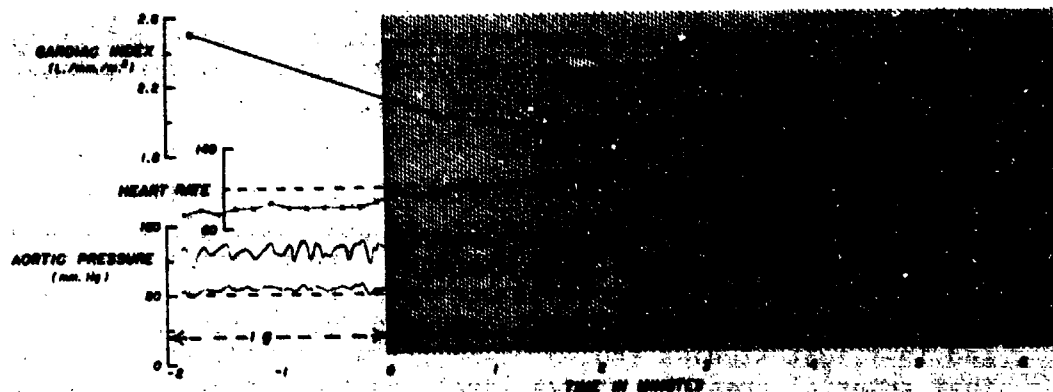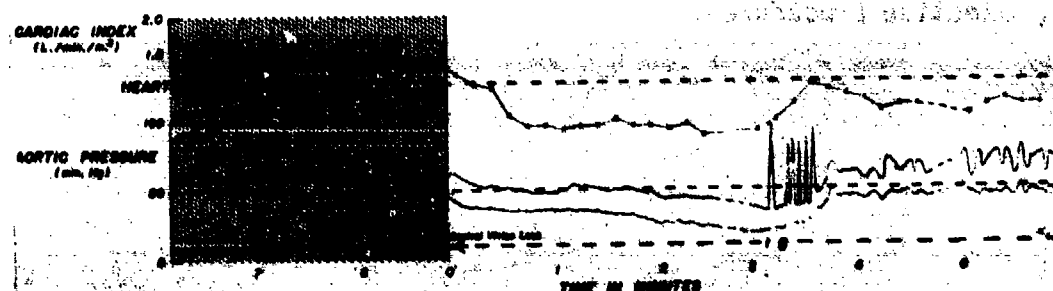

Figure 37. Plot of hemodynamic data during an 8½-min exposure to 3.5-G headward accelera- tion (stippled area), which was associated with development of a vasovagal type of reaction in a normal subject occurring toward the end of the exposure and continuing after its termination. Note the large, cyclic fluctuations in arterial pressure in the thoracic aorta that are usually seen prior to and during the exposure. After the onset of acceleration, the arterial pressure at heart level increased, as did the frequency and amplitude of the variations in this pressure. The intermittent determinations of cardiac output showed an initial decrease with the onset of acceleration, but no significant further systematic change was noted as the exposure was prolonged to 8½ min. During the seventh minute of the exposure, the variation in aortic pressure decreased, and an actual fall in both systolic and diastolic pressures occurred, beginning at minute eight. The heart rate failed to increase in response to this decrease in pressure, and the subject experienced loss of peripheral and then central vision prior to termination of the exposure. The pressure and heart rate con- tinued to fall after the centrifuge was stopped until 5 min after termination of the exposure, when the subject became nauseated to the point of retching. The pressure and heart rate then rose to normal values, and the normally pre- sent cyclic fluctuations in aortic pressure returned approximately 8 min after termination of the acceleration. (From Lindberg and Wood [70]).

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All went as planned during the first 8 min of the exposure. Then
quite suddenly the arterial pressure began to decrease followed by a slowing
of heart rate and loss of peripheral and central vision which terminated the
exposure. Somewhat alarmingly, the blood pressure and heart rate continued to
decrease to a systolic pressure of about 60 mmHg 5 min after the return to 1 G
at which point the subject was nauseated and retching, following which his
arterial pressure returned to more normal levels.

The Brooks, Farnborough, and Swedish human centrifuge groups have all
reported similar episodes which terminated prolonged simulated aerial combat or
plateau type G maneuvers. The fact that quite rapid, large decreases in cir-
culating blood volume invariably occur during high-G exposures (16,66,71)
coupled particularly, in inexperienced subjects with motion sickness, espe-
cially in student pilots, certainly sets the stage for vasovagal mediated
hypotensive episodes in flight and consequent, potentially disastrous losses
of consciousness at surprisingly low G levels.

A foolproof GLOC warning system, such as detection of zero arterial
pulsations at ear level to activate an automatic plane control takeover
system, could avoid most GLOC crashes. The potential efficacy of the use of
loss of arterial pulsations at ear level as a forewarning of potential loss of
vision and/or consciousness as has been documented both in human centrifuge
and in-flight studies (40,43,51-54,72,73) is indicated in Figure 38. Note
that arterial pressure at eye level and the ear pulse both decreased to zero
5 sec prior to loss of consciousness which would allow time for automated pro-
tective procedures.
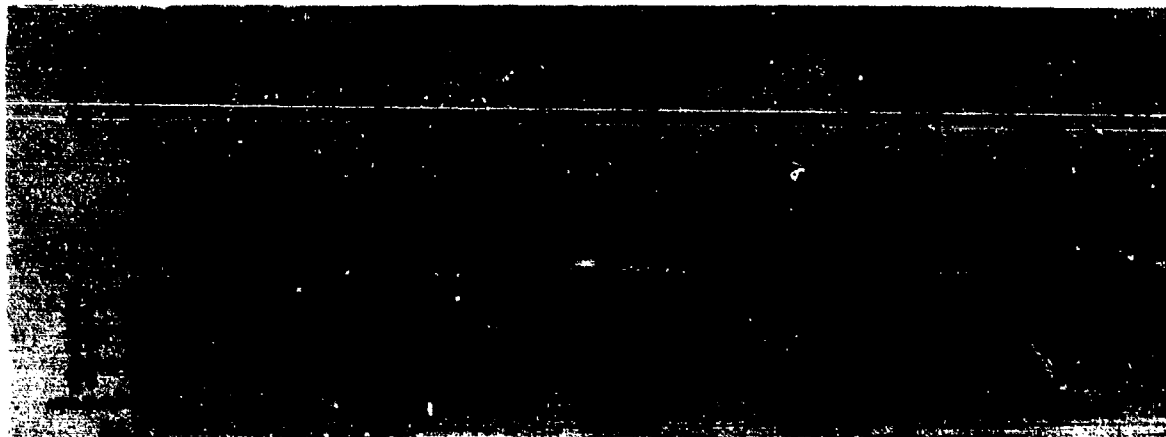
Figure 38. Effect of headward accelerations of increasing magnitude on:
arterial pressure at head level, subjective symptoms, and other
hemodynamic variables in a normal subject. PLR and CLR are
abbreviations for peripheral and central light responses, respec-
tively. The degrees of visual impairment are listed at the bottom
of each record (PLL indicates loss of peripheral vision; blackout
indicates loss of both peripheral and central vision.) Note (1) the
progressive increase in the magnitude of the alterations in the
various hemodynamic variables with increasing levels of accelera-
tion, (2) the correlation between arterial pressure at head level
and the changes in ear opacity, ear opacity pulse, heart rate, and
the occurrence of visual symptoms, and (3) that after detection
of loss of the ear opacity pulse (right-most panel), there would
be time to activate an automatic plane control take-over system
prior to or coincident with pilot loss of consciousness.

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These and other measures such as improved G-suit systems, partially supinating seats, etc., when perfected, could be implemented relatively soon. However, as long as use of upright sitting cockpit configurations persist, they will not eliminate incidents of GLOC in flight.

Personally, I am convinced that: First, full exploitation of the probable tactical advantages of piloted current and future super performance fighter planes, and second, practical elimination of GLOC in training and simulated combat maneuvers can only be achieved by use of a fully horizontal cockpit position combined with an optical-electronic all-directional pilot surveillance system. It seems apparent that these two items should be high-priority objectives of our Air Forces. If this is the case, improved G-suits, voluntary self-protective maneuvers and/or partially supinating seats are only stop-gap measures which leave the welfare of fighter pilots and their planes on a razor's edge. After all (Figure 39), Superman flies in the horizontal prone position. We should redesign our fighter plane cockpits and train our pilots so they can perform as Superman also.
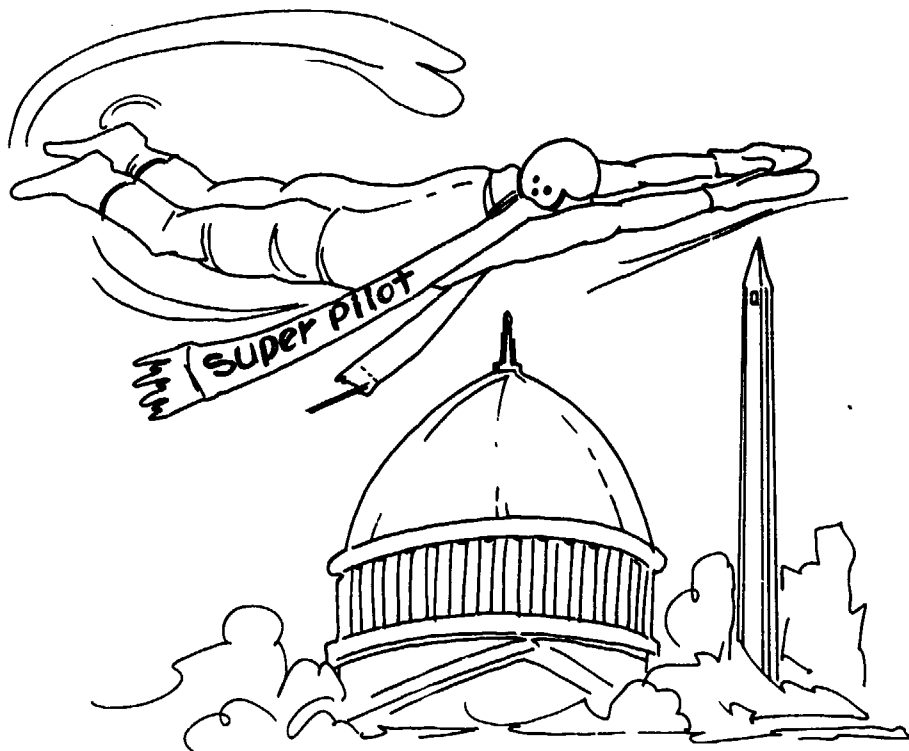

Figure 39. The physically and physiologically ideal body position for high-
            positive and negative-G maneuvers in flight (with permission
            [12]).

A carefully worked out fighter plane cockpit for a fully horizontal pilot position, preferably prone, is the only physically and physiologically certain and safest way to achieve these objectives.

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In this context, an article written a half century ago by a German expert on G tolerance, prior to World War II, reporting the relatively low G tolerance of subjects in the sitting position as contrasted to the very high tolerance when horizontal (74) is of interest. A statement of the essence of his concluding paragraph is as follows: "The cooperation of aircraft designers and manufacturers is, however, still missing. Their cooperation will be required to make this knowledge and these experimental results applicable to flying by proper redesign of the cockpit."

Initiation of the high expenditures of time, effort and expense required for perfection of a fully horizontal, omnidirectional pilot surveillance cockpit configuration will not be undertaken until convincing proof is at hand that significant tactical advantages will accompany pilot capabilities to sustain accelerations in the 7-15-G range for periods longer than the 5-7 sec GLOC latent period. Furthermore, such a large-scale cooperative effort will not be approved without pilot acceptance. This acceptance may eventually accrue because of experience-dictated realization by fighter pilots that, because of the severe fatigue and distracting characteristics of current G-suit/straining procedures, sustained-G combat maneuvers appreciably above the 7-G range cannot be flown effectively, consistently or safely in a conventional, upright or partially supinating cockpit configuration.

In the meantime, Dr. Harald von Beckh's studies and concern with this problem (75) should be kept in mind. His conclusions, which are paraphrased because of my belief that the horizontal prone position is physically and physiologically superior and psychologically preferable to the supine position, especially in combat are as follows: The urgency to provide fighter aircraft with horizontal se''s cannot be overemphasized. It is hoped that a potential adversary does not build such a seat first. A squadron equipped with a horizontal prone position cockpit with an optical-electronic omni-directional pilot surveillance system would have a spectacular advantage in air-combat situations, and could literally fly circles around their adversaries.

ACKNOWLEDGMENTS

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